

A Dissertation on

**“PREVALENCE OF HIV OCULAR MANIFESTATIONS IN RELATION
TO CD4 COUNT AT ART CENTRE, SMCH, CHENNAI”**

Submitted to the

THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.S. (Branch – III)

OPHTHALMOLOGY



GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMILNADU DR. M. G. R. UNIVERSITY,

CHENNAI, TAMILNADU

APRIL 2014

CERTIFICATE

This is to certify that the study entitled “**PREVALENCE OF HIV OCULAR MANIFESTATIONS IN RELATION TO CD4 COUNT AT ART CENTRE, SMCH, CHENNAI**” is the result of original work carried out by **Dr. Shubha.Raguram.K**, under my supervision and guidance at STANLEY MEDICAL COLLEGE, CHENNAI. The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in Ophthalmology**, course from May 2011 to April 2014 at Stanley Medical College, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled **“PREVALENCE OF HIV OCULAR MANIFESTATIONS IN RELATION TO CD4 COUNT AT ART CENTRE, SMCH, CHENNAI”** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Dr. K. Basker , M.S., D.O.**, Head Of The Department , Department Of Ophthlamology, Government Stanley Medical College and Hospital, Chennai- 600001.

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Title of the Work : Prevalence of HIV ocular manifestation in relation to CD4 Count at ART Centre, SMCH, Chennai

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Prevalence of HIV ocular manifestations in relation to CD4 count at ART centre ,
BY SHUBHA RAGURAM

INTRODUCTION

HIV-AIDS was discovered in the early 1970's. Amongst the various communicable diseases that have been well controlled with the advancing sciences , HIV- AIDS has been a major challenge¹. It poses a challenge not just to the doctors treating the disease but also to the patient subjected to societal stigma. AIDS with its multisystem manifestations calls for an increasing attention in understanding the disease and managing it and the associated opportunistic infections.

Eye with its adnexa is no exception to HIV infection. Ophthalmic presentations of HIV include microvasculopathy, neuro- ophthalmic disorders, neoplasms and opportunistic

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TOPIC:

PREVALENCE OF HIV OCULAR MANIFESTATION IN RELATION TO CD4 COUNT AT ART CENTRE, SMCH, CHENNAI..

KEYWORD: ocular HIV , HAART.

INTRODUCTION:

HIV;bane of new era has been a serious health issue since its discovery in the early 70's. Although with the current fall in the incidence globally and India; as per UN 2012 reports ;India is still the third largest nation with HIV population.

HIV manifests indirectly and it does so by decreasing the immunity especially CD4 cells,thereby leading to secondary infections. It affects all systems alike ;eye and its adnexa is no exception.

HIV ocular complications include minor opportunistic infection,tumor, inflammation, vasculopathy and retinopathy. It is important that these manifestations are detected irrespective of symptoms or history and treated at the earliest possible.

This study is done in order to find the prevalence of the various ocular manifestation of HIV ,its correlation to CD4 count and the role of HAART.

AIM

- To evaluate the prevalence of ocular manifestations in HIV seropositive patients, attending the ART centre, Stanley Medical College and Hospital.
- To evaluate how the manifestations correlate to the CD4 count.
- To evaluate the impact of HAART on ocular HIV manifestations.

METHODS AND MATERIAL

STUDY DESIGN:

Cross sectional study was undertaken on 100 HIV seropositive patient attending the ART centre with known CD4 count was included in the study. The study period was from February 2013 till completion of hundred patients.

INCLUSION CRITERIA

- HIV seropositive consented patients attending ART centre irrespective of ocular complaints, irrespective of treatment status.
- Any age group
- CD4 count obtained at the time of examination.

EXCLUSION CRITERIA

- Severely ill patients.
- CD4 count could not be obtained.
- Pre-existing systemic illnesses.

The importance of ocular examination in them was thoroughly explained in patients own language. Consent in patients own language was obtained from all those who were willing to take part in study. Any photographic records of the lesions were taken only if the patient consented for the same. Upon completion of data collection, statistical analysis was applied.

STATISTICAL ANALYSIS:

Chi square test was done for statistical analysis of the obtained data.

ROUTINE OCULAR EXAMINATION WAS DONE AS FOLLOWS:

- Ocular history.
- Ocular examination included:
 - Best corrected visual acuity. (Snellen's chart)
 - Slit lamp evaluation – Anterior and Posterior segment
 - Dilated fundus evaluation (0.8 % tropicamide with 5% phenylephrine) – 90 D
 - and 20 D
 - Neuro ophthalmological examination
 - Orbit
 - Tonometry for IOP evaluation
 - Schirmer's for dry eye evaluation
- Patient with suspected lesions were referred for complete systemic and laboratory work up.
- Serological investigations included TORCH titre, PCR assay for HZV, VZ.
- Other investigations were done as per clinical indication included CT brain/MRI brain.

OBSERVATION AND DISCUSSION:

The mean age of patients in our study was 38.14 +/- 11.84 which fell in line with mean age of the comparative studies. Majority of the patient prevalence in our study fell under the reproductive age group of 20-40 years. This pattern was similarly noticed in Biswas et al , Gururaj et al and Lamichhane G et al study. However our prevalence rate of 58 % was closely comparable to the Gururaj et al study which had 54 %. Also comparable with the Gururaj et al study was the decrease in prevalence with increase in age. Of special mention is the similarity in the prevalence of patients in the age group < 20 years.

Our study shows a higher male prevalence in comparison to females. The age gender distribution in our study was not significant, $p = 0.3$. This was also the finding in Lamichhane G et al study.

Sexual route was the most common mode of disease transmission, and this was observed in study by Biswas et al and Gururaj et al. Gururaj et al had 1 homosexual route of exposure. P value of 0.1274 signifies that the route of exposure has no impact the prevalence of the ocular manifestations. Majority of the patients in our study were observed in clinical stage I and II. The findings were similar in the Gururaj et al and Amare et al study.

It was observed in our study that all patients those who fell under the Clinical category III /IV had ocular HIV manifestation, and mostly of the opportunistic type. This was equally observed in the studies to which we compared to in the above table. With p value of 0.003 we can conclude that prevalence of ocular manifestation increases with the clinical staging of disease. And this is probably due to the increase in the

opportunistic infection in these stages. This was observed both in Gururaj et al and the Amare et al study as well. We observed an increased prevalence of ocular manifestations in the CD4 range of 200 – 500 cells/mm³. This was comparable to the Ethiopian study by Amare et al. P value in our study was 0.0235, which was significant. This significance was observed in all the above mentioned studies. Hence ocular manifestations is seen to increase significantly with decrease in CD4 count, especially in levels < 200 cells/mm³.

Blepharitis and conjunctival microvasculopathy was the commonest anterior segment findings observed in this study. The prevalence of both of which were significantly higher in comparison to the studies mentioned above. Kaposi sarcoma and conjunctival squamous cell carcinoma were nil, and were comparable to the two Indian studies mentioned above. These findings were present in the African studies. And this has been implicated due to the homosexual practises. We see differences in most of the anterior segment presentations in comparison to other Indian studies. This can be attributed to the geographical change in location of the study. The prevalence of HIV retinopathy, toxoplasmosis acute retinal necrosis and uveitis in our study was comparable to the prevalence in Biswas et al study. The same finding was not so with CMV retinitis. This limitation was observed probably due to the sample size in our study. Ocular TB and syphilis were not recorded in our study.

CONCLUSION

HIV infection is a problematic communicable disease present in our population, affecting commonly the reproductive age group. HIV manifests in the eye either directly in the form of viral load or causes low immunity thereby increasing chances for opportunistic infections. With the introduction of HAART, the life expectancy of the patients have significantly increased. However the ocular manifestations continue to present in innumerable forms. Not all patients with early HIV opportunistic infection present to us with ocular symptoms until unless the manifestation is severely blinding and irreversible. Most of the symptomatic patients are the ones with blepharitis and conjunctivitis, a non-blinding yet troublesome form of disease manifestations. The ART centres in India at present practice just the referral of patients for ophthalmological examination only when the patient develops ocular complaints. With the number of ocular findings observed, our study highlights the need for a routine ophthalmological screening of all HIV seropositive patients. We recommend a routine screening of HIV seropositive patients upon diagnosis, prior to starting ART therapy to obtain a baseline ocular status. Once the patient is started on HAART, he/she must undergo at-least a half yearly ocular examination. This is important for two reasons; one to look out for immune reconstitution syndromes, two to identify the ocular side effects of HAART. CD4 counts have to be strictly considered while monitoring these patients. It serves both as a risk factor as well as an indicator of opportunistic manifestations. A very important observation we made was the patients non-consenting to any detailed examination outside of the ART centre.

This was due to the stigma attached to the disease and the fear of being publically recognised as HIV seropositive. All this indicates a need for provision of ophthalmic setup in the ART centre. This can be made possible with adequate resources and trained ophthalmic personnel.

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PART I

INTRODUCTION

HIV-AIDS was discovered in the early 1970's. Amongst the various communicable diseases that have been well controlled with the advancing sciences, HIV- AIDS¹ has been a major challenge. It poses a challenge not just to the doctors treating the disease but also to the patient subjected to societal stigma. AIDS with its multisystem manifestations calls for an increasing attention in understanding the disease and managing it and the associated opportunistic infections.

Eye with its adnexa is no exception to HIV infection. Ophthalmic² presentations of HIV include microvasculopathy, neuro-ophthalmic disorders, neoplasms and opportunistic infections. Mainly seen and examined by the treating physician it becomes a necessity that all HIV infected patients undergo routine ophthalmic evaluations irrespective of symptoms for early identification of manifestations and prompt initiation of treatment to prevent visual loss.

This study was done to find the prevalence of the various ocular manifestation of HIV, its correlation to CD4 count and the role of HAART.

REVIEW OF LITERATURE

1. Ocular manifestation of HIV/AIDS and correlation with CD4+ cells count among adult HIV/AIDS patients in Jimma town, Ethiopia: a cross sectional study; Sisay Bekele, Yeshigeta Gelaw and Fasil Tessema.

Prevalence of HIV ocular manifestation was found to be lower than previous studies. They concluded this as probable result antiretroviral therapy. Low CD4 count is both risk factor and predictor for eye involvement in HIV.

2. Clinical study of ocular manifestations in HIV/AIDS and its correlation with CD4+T cells; Shivayogi Kusagur, Gururaj .K.J.

CD4+ cell counts and WHO clinical stage of HIV disease were concluded as important predictors of occurrence of ocular morbidity in HIV positive individuals.

3. Prevalence of HIV-associated ophthalmic disease among patients enrolling for antiretroviral treatment in India: A cross-sectional study ; Sophia Pathai, Alaka Deshpande, Clare Gilbert and Stephen D Lawn

One fifth Pre ART HIV individuals had ocular manifestations requiring treatment, and CMVR was the most common presentation. This highlights the need for screening HIV positive patients irrespective of ocular symptoms.

4. Pattern of Ocular Manifestation of HIV/ AIDS among Patients on HAART in ART Clinic of Gondar University Hospital, Northwest Ethiopia; Bemnet Amare, Fisseha Admassu, Yared Assefa, Beyene Moges, Jemal Ali and Afework Kassu.

The study demonstrates the need of eye care package in management of HIV/AIDS patients.

HISTORY

Theories of HIV origin and its epidemic have baffled many of us for ages. Simian immunodeficiency virus from monkeys³ spread⁴ into human community in various forms thereby mutating to Human immunodeficiency Virus (HIV). Homosexual⁵ practice along with social changes and urbanisation⁶ led into its amplification in human community.

The virus was identified and named⁷ lymphadenopathy associated virus in 1983 and renamed as Human lymphotropic virus-III⁸ in 1984. In the year 1986 both names were discarded and the name Human immunodeficiency virus was put into use. On retrospective analyses of all the preserved samples (blood, lymph node) of suspected deaths (clusters of immunosuppressive episodes) the first known case was a documented HIV1 infection from a man in Congo in the year 1959. Second documentation was from a woman in Congo in 1960. Since then number of patients positive for HIV have increased progressively without any permanent cure and increasing opportunist infections.

India is home to third largest population^{9,10} of people living with acquired immunodeficiency syndrome. The first reported case of HIV in India was by a female sex worker from Chennai in 1986. The first ocular lesion in HIV was reported from Sankara Nethralaya , Chennai in 1995¹¹.

GLOBAL SCENARIO

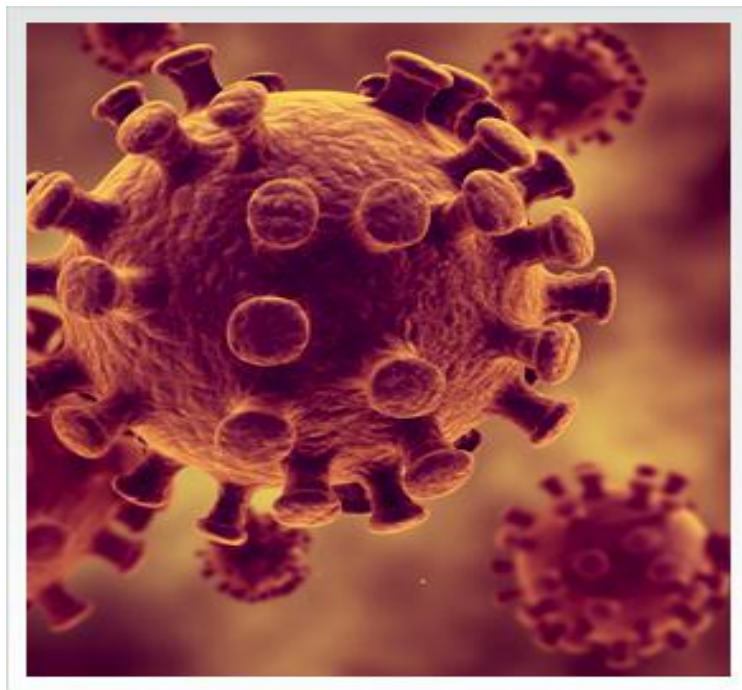
As on 2012, the estimated number of people living with HIV globally, is 35.3 (32.2–38.8) million. The number of new infection in 2001 was 3.4 million in comparison to 2.3 (1.9–2.7) million new HIV infections in 2012, showing a 33% decline in the number of new infections globally¹². The AIDS related deaths have drastically declined from 2.3 million in 2005 to 1.6 million in 2012, a direct result of increasing care and antiretroviral treatment. Even India is still the third largest nation with HIV population.

In India the new infection number fell from 2.74 lakhs cases in 2000 to 1.16 lakhs in 2011(57%). An impact of the various interventions under National AIDS Control Programme. Of the four high prevalence States in India includes Andhra Pradesh, Karnataka, Maharashtra and Tamil Nadu and they account for 53% of all HIV infected population in the country.

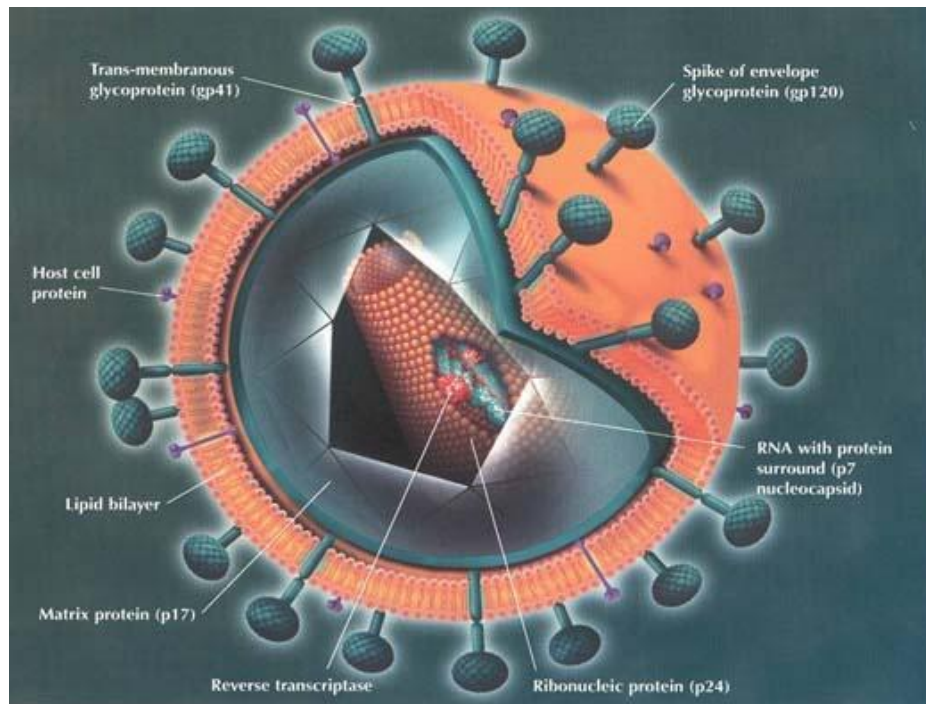
Prevalence of HIV in Tamilnadu is 0.33% as on 2012^{13,14}.

HUMAN IMMUNODEFICIENCY VIRUS

Human immunodeficiency virus¹⁵ (HIV) belongs to the genus lentivirus. Classified under the Retroviridae family it is a single stranded RNA virus. HIV causes acquired immunodeficiency syndrome (AIDS), a condition characterised by progressive failure of the immune system.



MICROSCOPIC STRUCTURE OF HIV VIRION



Shape¹⁶: spherical enveloped virus

Size: 90-120nm

Nucleocapsid: has an outer icosahedral shell and inner cone shaped core that which encloses ribonucleoproteins.

Genome: diploid composed of two identical single stranded positive sense RNA copies. Bound to viral RNA is the reverse transcriptase enzyme. 10⁻¹⁵ in numbers. Also contains integrase enzymes and transfer RNA.

VIRAL GENES AND ANTIGENS:

STRUCTURAL GENES:

These are the gag, pol, env; the products of which are the viral antigens¹⁷.

1. gag: precursor proteins that determine the core and the shell of the virus.(p15, p18, and p24).

p24 helps in disease detection and indications re-exacerbation.

2. env: determines synthesis of envelope glycoprotein gp120 and gp 41.

gp120- forms surface spikes¹⁷.

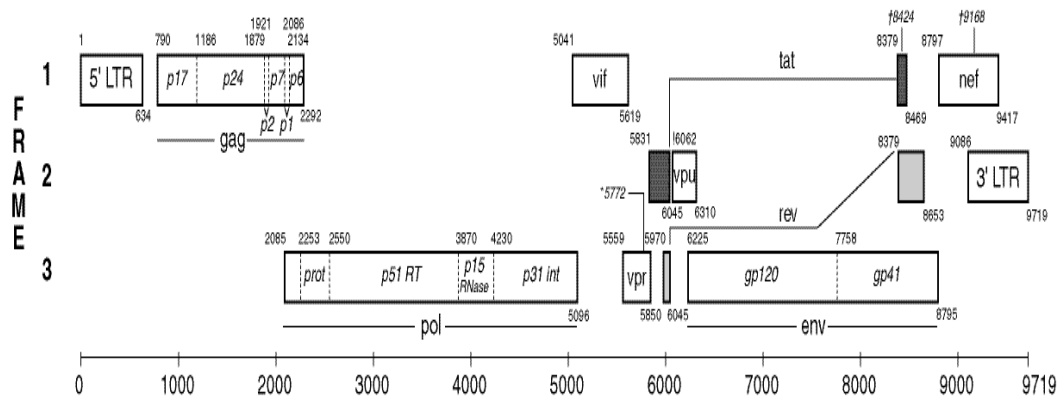
gp 41 -forms the trans membrane anchoring
protein¹⁸.

3. pol :codes for polymerase reverse transcriptase ,protease and endonuclease

NON-STRUCTURAL AND REGULATORY GENE:

1. tat (trans activating gene): enhances expression of all viral genes.
2. nef (negative factor gene) : down regulates viral replication.
3. rev (regulator of virus gene): enhances expression of structural proteins.
4. vif (viral infectivity factor gene): influencing infectivity of viral particles.
5. vpu (HIV1) and vpx(HIV2) : enhancing maturation and release of progeny virus

6. vpr stimulating promoter region of the virus.
7. LTR (long terminal repeat)¹⁹ sequences, one at either end, containing sequences giving promoter, enhancer and integration signals.



ANTIGENIC VARIATIONS:

HIV has been characterized into HIV-1 and HIV-2²⁰.

HIV1: responsible for majority of infection. It is more virulent of the two. Three main are Group M (majority of cases), Group O and Group N.

HIV2: largely confined to West Africa because of its low virulence.

VIRUS STABILITY:

HIV is thermolabile²¹, being inactivated in 10 minutes at 60 °C and in seconds at 100 °C. In dried blood it may survive for up to seven days. It withstands lyophilisation.

HIV is inactivated in 10 min by treatment with 50% ethanol, 35% isopropanol, 0.5% lysol, 0.5% paraformaldehyde, 0.3% hydrogen peroxide, 10% household bleach. Treatment of contaminated articles is with 2% solution of glutaraldehyde²².

CD4 AND HIV

CD is an abbreviation of the cluster of differentiation /cluster of designation²³. They are a defined sub classification of cellular surface receptors (> 250 in number), also called epitope; which identify cell type and stage of differentiation. These receptors coat the surfaces of the B and T lymphocytes and are recognized by antibodies. Of all the epitopes classified CD4 receptor present on the T lymphocytes has been in limelight for its highest association with HIV pathogenesis.

CD4 is a membrane glycoprotein present on precursor T cells and also on a few mature T cells. Being a helper cell, when activated in presence of an infecting agent (antigen) triggers a cascading event thereby activating the co immune cells especially the killer T cells (CD 8) thereby eliminating the antigen²⁴.

HIV gains its entry into host immune system by attaching itself to CD4 surface receptor. Once infected by HIV there is a progressive depletion of both HIV infected and un-infected immune cells. The infected cells undergo programmed cell death by Fas mediated apoptosis²⁵. The un-infected bystander immune cells undergo leaking HIV protein induced cell death, activation induced cell death (AICD), and cytokine mediated cell death. All the factors stated above results in failure of immune system.

CD4+ T cell count in HIV infected individuals categorises the systemic/opportunistic manifestations. CD4 cell counting includes various techniques of which flow cytometry²⁶ is essentially used. CD4 values ranging from 500-1200 cells/mm³ is considered normal range for HIV infected person. A fall below 500cells/mm³ warrants frequent monitoring, and a fall below 350cells/mm³ necessitates start of antiretroviral therapy.

However a child is born with a very high level of CD4 (several thousands) and this cannot be used in monitoring disease. Therefore CD4 percentage in relation to other immune cells is used²⁷.

CD4% of 12-15% = <200 cells/mm³,

CD4% of 29% = > 500 cells/mm³,

CD4% of about 40% = HIV negative³⁴.

For treatment purposes in HIV infected child World Health Organisation recommends the following²⁸:

AGE	Infants and children <24 months of age	≥24 months of age to 59 months of age	Five years of age or Older
%CD4+	Absolute lymphopenia	≤25	NA
Absolute CD4 count	Absolute lymphopenia	≤750 cells/mm ³	≤350 cells/mm ³ (As in adults)

HIV TRANSMISSION

TYPES OF EXPOSURE	CHANCE OF INFECTION PER EXPOSURE
Sexual intercourse: anal , vaginal , oral	0.1 – 1.0 %
Blood and blood products, blood transfusion	> 90%
Tissue and organ donation: semen , cornea, bone marrow, kidney etc	50 -90%
Injections and injuries: injection with unsterile syringes, drug addicts shared needles, needle stick injury , surgical wounds	0.5 -1.0%
Mother to baby :trans-placental , at birth , after birth breast milk	30%

SEXUAL TRANSMISSION:

Predominant mode of transmission. Risk of transmission increases with multiple sexual partners²⁹. People indulged in riskful sexual practices. These people and their partners should be counselled regarding safer sex methods.

BLOOD AND BLOOD PRODUCTS:

Second important mode of transmission³⁰. Major mode of transmission prior to recognition of HIV. Now with blood screening made mandatory the risk is reduced considerably.

ORGAN TRANSPLANTATION:

Organs are now screened for HIV and risk of transmission has been drastically reduced. This is applicable for semen, cornea, bone marrow, kidney and other organs.

INJECTIONS:

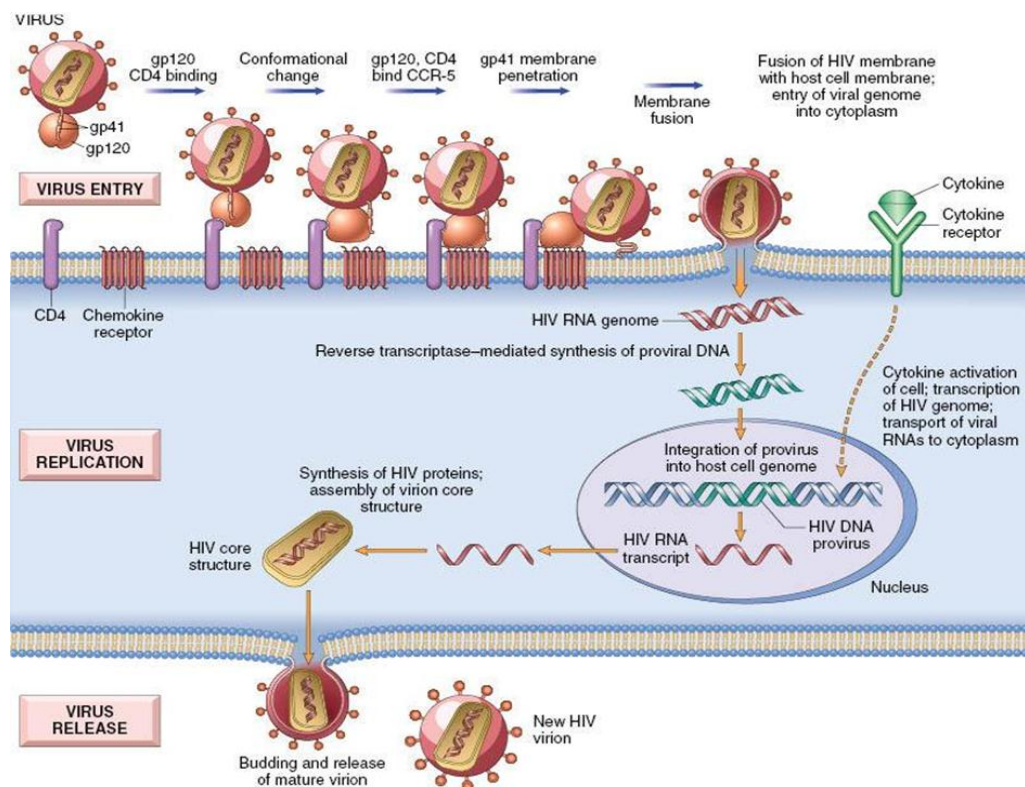
Relevant in intravenous drug abusers who share needles³¹. Also in medical and health professional the danger of needle stick injury persists to a great extent, though the chance of infection is much less.

MOTHER TO CHILD:

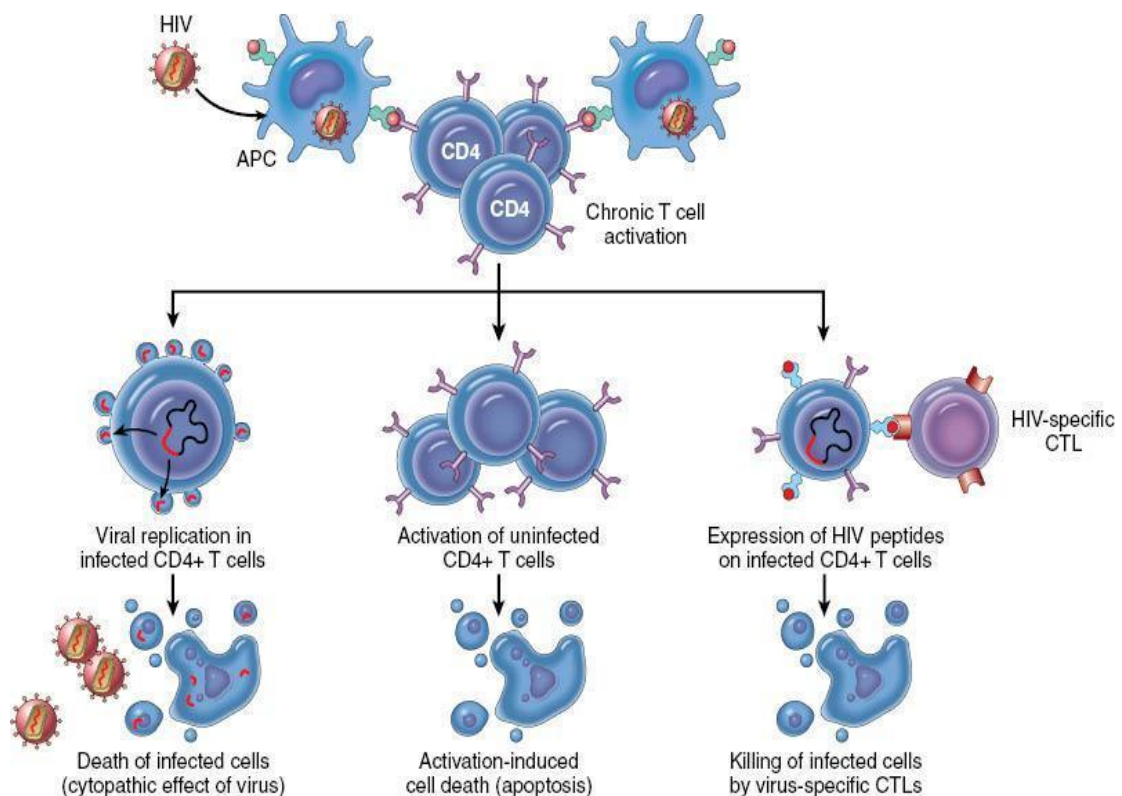
Child can be infected transplacentally³², during birth or with breast feeding. 30% of the infection occurs in utero and 70% during or after delivery.

PATHOGENESIS

HIV infects the host system, integrates its provirus into host cell genome³³, activates viral replication thereby producing and releasing infectious virus. Gp120 of the virus binds to the CD4 receptor present on the T lymphocytes. The co receptors³⁴ namely CCR5 and CXCR4 are equally important for virus entry into the cell for R5 and X5 strain of HIV respectively. Gp120 binding brings about conformational changes in Gp41, thereby allowing membrane penetration and fusion for viral membrane with host cell membrane.



This is followed by entry of viral genome into cytoplasm. Once in the host cytoplasm HIV RNA undergoes reverse transcriptase mediated proviral DNA synthesis. HIV proviral DNA now integrates with host genome, and undergoes transcription and synthesis of HIV protein. Virion core structure assembly takes place. These core structures bind to host cell membrane followed by budding and release of mature virion. Multiple new virions are released in this process. These new virions in turn infect new host cells and the entire cycle of replication and release continues. This vicious cycle ultimately leads to irreparable deterioration of immune system.



HIV CASE DEFINITION AND CLASSIFICATION

PROVISIONAL DIAGNOSIS FOR AIDS, WHO: (1986)³⁶

Presence of at least two major signs along with one minor sign, from those listed below

MAJOR SIGN
Weight loss greater than 10% of body weight
Fever for more than one month, intermittent or continuous
Chronic diarrhoea for more than one month, intermittent or continuous
MINOR SIGN
Persistent cough for more than one month
General skin manifestation
Recurrent herpes zoster
Oropharyngeal candidiasis
Chronic progressive and disseminated herpes simplex infection
Generalised lymphadenopathy

CDC CLASSIFICATION SYSTEM FOR HIV DISEASE: (1986)

Group I: acute infection

Group II: asymptomatic infection

Group III: persistent generalised lymphadenopathy

Group IV: other diseases

- Subgroup A constitutional disease
- Subgroup B neurologic disease
- Subgroup C secondary infectious disease
- Subgroup D secondary cancers
- Subgroup E other conditions

Revised CDC classification of 1995

Stage 1 (HIV infection)	CD 4 t lymphocyte count greater than or equal to 500 cells/MI
Stage 2 (HIV infection)	CD4 T lymphocyte count of 200-499 cells/uL
Stage 3 (AIDS)	CD4 T lymphocyte count of less than 200 cells/uL

WHO STAGING:

HIV-ASSOCIATED IMMUNODEFICIENCY	AGE RELATED CD4 VALUES IN %			
	<11 months	12-35 months	36-59 months	>5 years
None or not significant	>35	>30	>25	>500
Mild	30-35	25-30	20-25	350-499
Advanced	25-29	20-24	15-19	200-349
Severe	<25	<20	<15	<200 or 15%

LABORATORY DIAGNOSIS

Test includes for immunodeficiency as well as specific test for HIV.

Specific test for HIV³⁷

1. Antigen detection: p24 is the earliest virus marker to occur and detected by ELISA.
2. Polymerase chain reaction: sensitive and specific test. DNA PCR and RNA PCR are the tests performed. The tests are complex and costly and indicated only when other tests do not give definitive results.
3. Virus isolation: From peripheral lymphocytes. Patient's lymphocytes co cultivated with uninfected lymphocytes in presence of interleukin - 249.
4. Antibody detection: two type of tests- screening and confirmatory

ELISA: screening test. Simple, inexpensive but false positives are common.

WESTERN BLOT: it's a gold standard confirmatory test.

Immunological tests:

1. Total leucocyte and lymphocyte count
2. CD4 cell count
3. Platelet count
4. Raised IgG and IgA level
5. Lymph node biopsy

ANTIRETROVIRAL THERAPY

Standard antiretroviral therapy (ART)³⁸ consists of the combination of at least three antiretroviral (ARV) drugs to maximally suppress the HIV virus and stop the progression of HIV disease.

Various antiretroviral drugs approved for therapeutic use in India:

NRTI	NNRTI	Protease inhibitor	Entry inhibitors	Integrase inhibitors
Zidovudine	Nevirapine	Saquinavir	Enfuvitride	Raltegravir
Stavudine	Efavirenz	Indinavir	Maraviroc	
Lamivudine	Etravirenz	Ritonavir		
Abacavir	Delavirdine	Nelfinavir		
Didanosine		Lopinavir		
Tenofovir		Atazanavir		
Dideoxycytidine		Darunavir		
		Fos-Amprenavir		
		Tipranavir		

NRTI : nucleoside reverse transcriptase inhibitor;

NNRTI: non-nucleoside reverse transcriptase inhibitor

Highly active antiretroviral therapy (HAART) is the combination of two or more groups of drugs that acts at different level of viral replication thereby increasing patient compliance and also decreasing chances of drug resistance. HAART is initiated when the CD4 count <350/uL, the combination depending on patient health status and other co morbidities.

Most current HAART regimens consist of three (3) drugs: 2 NRTIs + a PI/NNRTI/II

OCULAR COMPLICATIONS OF ANTIRETROVIRAL THERAPY

Given below are the common drug used in our set up and its side effects;

NRTI	ADVERSE EFFECT
Zidovudine	Amblyopia, macular edema, photophobia
Stavudine	-
Lamivudine	Red itchy eye
Abacavir	Red itchy eye
Tenofovir	-
didanosine	Retinal changes, optic neuritis

NNRTI	ADVERSE EFFECT
Nevirapine	Red itchy eye

PROTEASE INHIBITOR	ADVERSE EFFECT
Saquinavir	blepharitis, dry eye syndrome, eye irritation, xerophthalmia, cytomegalovirus retinitis
Indinavir	Ocular pain , orbital edema
Ritonavir	Iritis , visual field defects, abnormal EOG
Nelfinavir	Acute iritis

HIV IN OPHTHALMOLOGY

CORRELATION OF CD4 T LYMPHOCYTE COUNT AND OCULAR LESION³⁹:

Stage	Seroconversion ⁶³	Early infection	Intermediate infection	Late infection
CD4+	1000	500-1000	200-500	<200
External eye	Inflamed conjunctiva, Dry eye	Allergic conjunctivitis	Dry eye, Blepharitis, Bacterial and follicular conjunctivitis, Kaposi's sarcoma, Molluscum contagiosum,	Opportunistic infections and tumours affecting all ocular structures
Anterior segment		Reiters syndrome Intermediate uveitis	Herpes simplex, Herpes zoster	
Posterior segment		HIV retinopathy Retinal vasculitis	HIV retinopathy, Tuberculous uveitis	
Neuro ophthalmic	Headache Retro orbital pain	Optic neuropathy	Aspergillosis	

SITE WISE OCULAR LESION IN AIDS:

SITE	LESIONS
ADNEXA	Herpes zoster ophthalmicus Kaposi sarcoma Molluscum contagiosum Eyelid infections Allergic or infective conjunctivitis
ANTERIOR SEGMENT	Dry eye Infective keratitis Uveitis Microvasculopathy
POSTERIOR SEGMENT	HIV retinopathy CMV retinitis Progressive outer retinal necrosis Acute retinal necrosis Fungal endophthalmitis Toxoplasma retinochoroiditis Ocular syphilis Mycobacterium infection
ORBIT	Orbital cellulitis Burkitts lymphoma
NEURO OPHTHAL	Cranial nerve palsies Papilledema Optic neuropathy

DESCRIPTION OF HIV OCULAR LESIONS

EYELID INFECTIONS

Blepharitis and meibominitis are common adnexal presentations and require prompt treatment which includes lid hygiene, hot fomentation and support antibiotics.

MOLLUSCUM CONTAGIOSUM

HIV infected patients have an extensive, coalescing lesions and these recur on treatment. Treatment includes cautery- electrical or chemical, surgical excision.

KAPOSI SARCOMA (eyelid and conjunctiva)

A vascular lesion, reddish purple in colour arising from the lid and or conjunctiva. Usually said to be associated with herpes virus 8 infections. Kaposi sarcoma in HIV has not been reported in Indian context.

CONJUNCTIVAL MICROVASCULOPATHY

AIDS virus is said to directly attack the vascular wall leading to its dilation/ segmentations, and cock-screwing.

HERPES ZOSTER OPHTHALMICUS

5- 15% of HIV population will have varicella zoster infection associated with keratitis, uveitis, scleritis, and retinitis. Atypical presentation with bilateral involvement is commonly seen. Treatment is with acyclovir given intravenous and oral for maintenance.

INFECTIVE KERATITIS

HIV individuals are easily susceptible to corneal infection from bacterial, viral, fungal and protozoal.

UVEITIS

Unexplained uveitis in an individual should prompt investigations for HIV. Uveitis in HIV-infected patient should prompt a search for an underlying infection, including tuberculosis, syphilis, histoplasmosis, coccidioidomycosis, and toxoplasmosis.

HIV RETINOPATHY

It is a non-infectious microvascular disorder of the retina in HIV infection. With cotton wool spots, microaneurysms retinal haemorrhages and capillary non perfusion this almost mimics diabetic retina. Therefore it becomes a necessity to rule out HIV in unexplained vascular occlusive diseases.

The virus is said to deposit on the vascular endothelium thereby inciting an antigen antibody mediated immune reaction leading to vascular necrosis and secondary ischemia. Cotton wool spots are more commonly seen than haemorrhages due to the above mentioned reason.

Hemorrhages are seen less commonly than cotton-wool spots. They may involve both the nerve fiber layer and the deeper retina and may appear as flame-shaped, dot, or blot hemorrhages.

CYTOMEGALOVIRUS RETINITIS

Is the most common opportunistic infection in HIV. Cytomegalovirus is a DNA herpes group virus occurring in advanced HIV disease. This DNA virus invades the retinal cells causing retinal necrosis. Manifests either in indolent or fulminant form. The indolent variety has granular white retinal deposits, has minimal or no vasculitis and no retinal hemorrhage. The fulminant type presents with retinal deposits, vasculitis and haemorrhages. Both varieties begin in the periphery and spread to centre along the vascular arcade leading to triangular / wedge zones. Vitritis if at all will be minimal. Frosted branch angiitis in another mode of presentation. With eventual loss of retinal pigment epithelium the underlying choroidal vasculature becomes prominent.

Treatment includes both systemic and intravitreal injections of gancyclovir. Other drugs include valcyclovir, foscarnet. Administer till lesion heals. Retinal detachment can be a major challenge while treating CMV retinitis.

ACUTE RETINAL NECROSIS

Rapidly progressing bilateral viral uveitis in immunocompromised patient caused most commonly by Varicella followed by herpes or CMV. bilateral involvement

is common with complications like retinal detachment and proliferative vitreoretinopathy. Ganciclovir /foscarnet, with adjunctive high-dose intravenous acyclovir (15 mg per kilogram every 8 hours) is the treatment of choice.

TOXOPLASMA RETINOCHOROIDITIS

Protozoan parasite which exists in cyst and trophozoites form. Though the infection occurs in early in extremely immunocompromised person, the ocular manifestation occurs usually on immune recovery on administration of HAART. This is because the immune mediated reaction is directed towards the cyst which is severe. In HIV, toxoplasmosis is characterised by multifocal retinochoroidal fluffy lesions involving entire retina with severe vitritis. Degree of haemorrhage is less in comparison to CMV retinitis. Fluorescein angiography differentiates toxoplasmosis from CMV retinitis by active leakages.

Serological testing for toxoplasma in HIV is unreliable. They either show low IgG titres or are unable to distinguish old from new lesions. Negative IgG titre however makes it unlikely for toxoplasma infection to be present.

Standard dosage treatment with pyrimethamine, sulfadiazine or clindamycin is said to be sufficient. Treatment is continued till the normal range of CD4 is achieved for HIV patients.

PNEUMOCYSTIS CHOROIDITIS:

Pneumocystis jiroveci (PCP), is a fungus showing protozoal characteristics. It presents as multiple yellow white choroidal lesions, generally round and of variable size. It eventually leads to chorioidal necrosis. The earliest evidence of PCP retinal infection was seen in 1982, where the histopathological report showed evidence of PCP in ganglion and plexiform layer of retina. In 1987 histopathologic examination revealed areas of choroidal thickening and exudate which harbored the characteristic cysts of P jiroveci in HIV patients. Treatment will be same as for respiratory PCP infection.

SYPHILIS

Syphilitic presentations include perivasculitis, hemorrhages, chorioretinitis , panuveitis and papillitis. These patients manifest symptomatic neurosyphilis. Lumbar puncture is the current recommendation for ocular syphilis in HIV to rule out neurological involvement. The serum fluorescent treponemal antibody absorption test (FTA-ABS) is the most reliable laboratory studies for diagnosing ocular syphilis. Syphilis runs a rapid aggressive course in HIV-infected patients than in immunocompetent individuals.

OPTIC DISC

Optic disc involvement includes papilledema as a result of intracranial space occupying opportunistic lesions, optic neuritis or neuropathy.

Optic neuritis in HIV is either a primary HIV manifestation or a secondary opportunistic manifestation. HIV is said to have a direct cytotoxic effect on axonal cells causing inflammation of optic nerve and death. Eventually optic atrophy ensues³⁹.

TREATMENT OF OCULAR HIV MANIFESTATIONS

LID INFECTIONS:

Lid hygiene is of utmost importance. Antibiotics ointment and oral antibiotics are necessary. Also strict adherence to antiretroviral therapy helps to maintain good immunity.

MOLLUSCUM CONTAGIOSUM:

Strict ART follow up. Topical trichloroacetic acid can be applied. Surgically curettage, excision or cryotherapy can be done.

CONJUNCTIVITIS:

Infective conjunctivitis needs to be proved on gram stain and culture, and treated accordingly.

If allergic the drug causing should be replaced and symptomatic topical medication like lubricating drops applied.

CONJUNCTIVAL MICROVASCULOPATHY:

This finding has no treatment.

HERPES ZOSTER OPHTHALMICUS:

Acyclovir 10 milligram per kilogram of body weight administered IV every 8 hours for 1 week. Maintenance dose of oral acivir 800mg, 3-5 times a day indefinite.

On follow up patient had to be treated for post herpetic neuralgia if sny.

Alternatives include valacyclovir.

VIRAL KERATITIS:

If the cause is varicella zoster, oral acivir 800mg 5 times a day is given. For resistant cases IV foscarnet has been useful. Topical trifluridine 1 percent is also seen to be helpful in controlling varicella keratitis.

If the cause is herpes zoster; same treatment as for varicella. Topical application of antiviral medication is most indicated in herpes keratitis as it tends to recur more frequently in HIV AIDS patients.

FUNGAL AND BACTERIAL KERATITIS:

Treatment depends on the culture sensitivity and grams stain reports. Strict adherence to both ocular medication and antiretroviral therapy ensures a quick healing. These patients go in for perforations easily and needs a strict follow up.

UVEITIS:

Usually drug induced, treatment consists of topical steroids tapered over weeks till resolution.

GLAUCOMA:

Usually of the inflammatory component, the underlying cause has to be treated first and also topical anti glaucoma medication have to be administered taking care to avoid prostaglandin analogue.

HIV RETINOPATHY:

Immune status needs to be evaluated and patient followed up every 3 months for dilated fundus examination.

CMV RETINITIS:

Ganciclovir 5mg/kg IV, 12th hourly for 3 weeks. Followed by maintenance doses of 5mg/kg/day is given twice weekly until lesion is inactive.

Alternate drugs include foscarnet and valganciclovir.

Immune status has to be improved with strict adherence to HAART. If the patient has never been on HAART prior to the occurrence, regime can be postponed to prevent immune recovery uveitis.

Patient needs to be educated about the recurrence and must be advised for a serial follow for dilated fundus examination.

TOXOPLASMOSIS:

Trimethoprim/ sulfamethoxazole 400mg administered per oral twice a day for 4 to 6 weeks.

Patients with very low immunity have to be given at least one drug life time or till immune status improves.

ACUTE RETINAL NECROSIS/ PROGRESSIVE OUTER RETINAL NECROSIS:

Intravenous 15mg/kg acyclovir is given 8th hourly. Foscarnet or Valcyclovir could be prescribed alternatively. Patient needs to be monitored for drug induced complications. Usually progresses rapidly.

MULTIPLE CRANIAL NERVE PALSY:

Find the underlying cause and treat.

OPTIC NEURITIS:

Underlying cause has to be found and treated.

OCULAR TUBERCULOSIS:

Rifampicin 500mg/kg, isoniazid 5mg/kg, pyremethamine 30 mg/ kg, ethambutol 15mg /kg; for a duration of two months and followed by rifampicin and isoniazid for remaining 4 to 7 months depending on patient's response to medication.⁴⁰

PART II

AIM

- To evaluate the prevalence of ocular manifestations in HIV seropositive patients, attending the ART centre, Stanley Medical College and Hospital.
- To evaluate how the manifestations correlate to the CD4 count.
- To evaluate the impact of HAART on ocular HIV manifestations.

METHODS AND MATERIAL

STUDY DESIGN:

- Cross sectional study was undertaken on 100 HIV seropositive patient attending the ART centre with known CD4 count was included in the study.
- The study period was from February 2013 till completion of hundred patients.

INCLUSION CRITERIA

- HIV seropositive consented patients attending ART centre irrespective of ocular complaints, irrespective of treatment status.
- Any age group
- CD4 count obtained at the time of examination.

EXCLUSION CRITERIA

- Severely ill patients.
- CD4 count could not be obtained.
- Pre-existing systemic illnesses.

- The importance of ocular examination in them was thoroughly explained in patients own language.
- Consent in patients own language was obtained from all those who were willing to take part in study.
- Any photographic records of the lesions were taken only if the patient consented for the same.
- Upon completion of data collection, statistical analysis was applied.

STATISTICAL ANALYSIS:

- Chi square test was done for statistical analysis of the obtained data.

ROUTINE OCULAR EXAMINATION WAS DONE AS FOLLOWS:

➤ Ocular history.

➤ Ocular examination included:

Best corrected visual acuity. (Snellen's chart)

Slit lamp evaluation – Adnexa and Anterior segment

Dilated fundus evaluation(0.8 % tropicamide + 5% phenylephrine)90 D

and 20 D

Neuro ophthalmological examination

Orbit

Tonometry for IOP evaluation

Schirmer's for dry eye evaluation

➤ Patient with suspected lesions were referred for complete systemic and laboratory work up.

➤ Serological investigations included TORCH titre, PCR assay for HZV, VZ.

➤ Other investigations were done as per clinical indication included CT brain/ MRI brain.

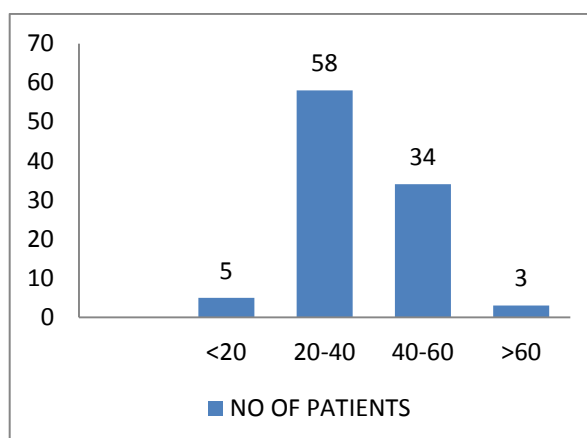
OBSERVATIONS

This study includes 100 consented HIV seropositive patients, irrespective of age or gender, provided CD4 count was available at the time of ocular examination.

AGE WISE DISTRIBUTION: N = 100

Table 1

AGE IN YEARS	NO OF PATIENTS
<20	5
20-40	58
40-60	34
>60	3



- The overall average age of the study population was 38.14 years.
- The reproductive age group of twenty to forty years formed the major part of our study, with 58 patients falling under this group.

- Of these fifty-eight patients, forty-three fall under age group of thirty-one to forty years.

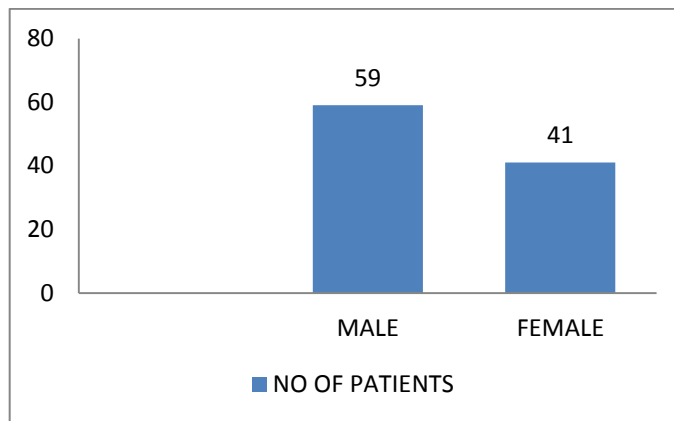
- In the age group less than twenty years, 5 children were observed. The average age is 9 years.

GENDER DISTRIBUTION: N= 100

With no attempt to maintain an equal count of the gender population, we observed 59% males and 41% females.

Table 2

GENDER	NO OF PATIENTS
MALE	59
FEMALE	41



AGE DISTRIBUTION OF GENDER WAS AS OBSERVED BELOW:

Table 3

AGE	GENDER	
	MALE	FEMALE
<20	2	3
20-40	32	26
40-60	22	12
>60	3	0

➤ The age of the male patients ranged from thirteen to seventy-seven years.

Whereas age of female patients ranged from four to fifty-seven years.

- The mean age for the fifty nine male patients is 39.7 years. And for the forty-one female patients is 35.9 years.

Table 4

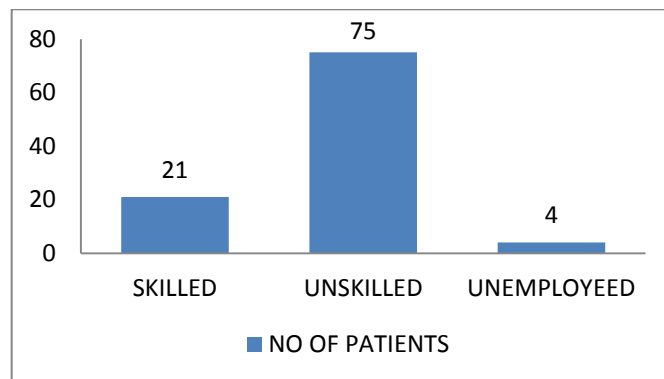
GENDER	NO OF PATIENTS	AVERAGE AGE (MEAN)	STD DEVIATION FOR AGE	P value
MALE	59	39.7	11.75	0.303079
FEMALE	41	35.9	11.27	

- Upon statistically validating the age – gender distribution using chi square test, we arrived upon a p value of 0.303079.
- Hence this shows that the given gender distribution for age did not affect the overall study.

OCCUPATION: N = 100

Table 5

OCCUPATION	NO OF PATIENTS
SKILLED	17
UNSKILLED	79
UNEMPLOYED	4
TOTAL	100



AGE WISE OCCUPATION DISTRIBUTION:

Table 6

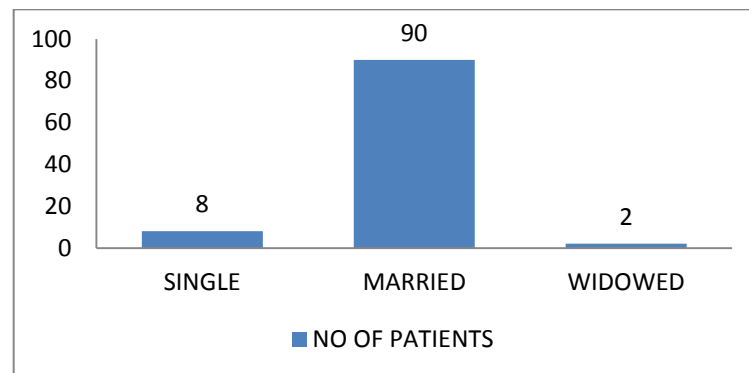
AGE	OCCUPATION		
	SKILLED	UNSKILLED	UNEMPLOYED
<20	0	5	0
20-40	11	46	1
40-60	6	27	1
>60	0	1	2
Chi Test =0.000015, Df= 6			
P value= 0			

- 79 Unskilled workers formed a major part of our study, with 46 of them falling under the reproductive age group. Majority of these unskilled workers were drivers.
- The 5 children were included under the unskilled group.
- The 11 skilled workers, including businessmen also were of the reproductive age group.
- There is a high prevalence rate of disease amongst the unskilled workers and is statistically proved.

MARITAL STATUS: N= 100

Table 7

MARITAL STATUS	NO OF PATIENTS
SINGLE	8
MARRIED	90
WIDOWED	2
TOTAL	100

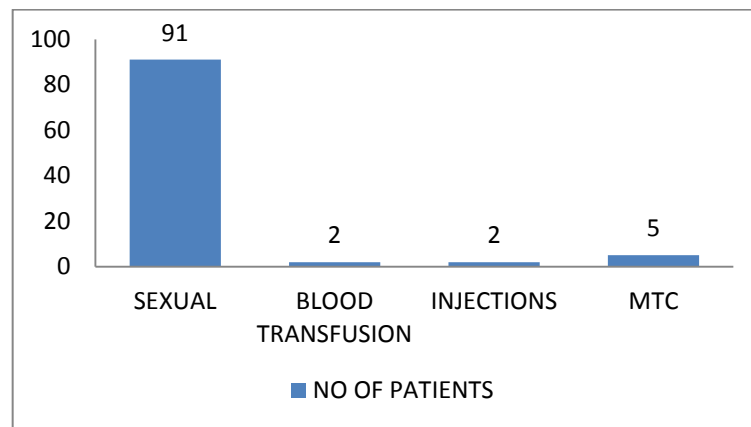


- 90 out of the 100 patients in our study were married and contracted disease either from multiple sexual partners or from the exposed spouse.
- Interestingly 1 widow was diagnosed with the disease following death of the male spouse from undiagnosed illness.
- The single group of 8 people included 5 children and 3 unmarried adult males.

MODE OF DISEASE EXPOSURE: N = 100

Table 8

MODE OF DISEASE EXPOSURE	NO OF PATIENTS
SEXUAL	91
BLOOD TRANSFUSION	2
INJECTIONS	2
MTC	5
TOTAL	100



- We observed that Sexual exposure was the most common form of disease transmission, with all 91 patients having had heterosexual exposure.
- 5 children had vertical transmission of the disease from the exposed mother (mother to child transmission/ MTC).
- 4 patients amongst the reproductive age group had non sexual exposure. Two of which had history of blood transfusion in the recent past, and 2 had high risk IV drug abuse behaviour with needle sharing.

WHO CLINICAL STAGE: N = 100

Table 9

WHO CLINICAL STAGE	NO OF PATIENTS
I	73
II	7
III	10
IV	10
TOTAL	100

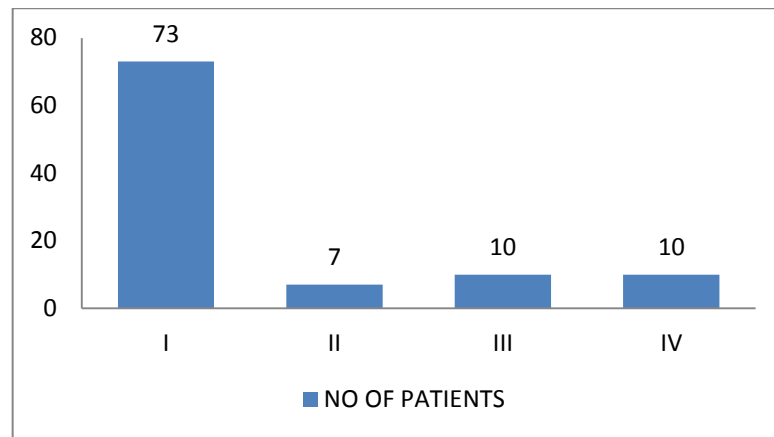


Table 10

WHO CLINICAL STAGE	OCULAR LESION		TOTAL
	PRESENT	ABSENT	
I	54	19	73
II	7	0	7
III	8	2	10
IV	9	1	10
TOTAL	78	22	100
P Value = 0.003			

- WHO clinical stage I consisted of 73 patients, indicating that the patients were asymptomatic or had acute flu like symptoms on disease detection.

- The 20 patients from clinical stage III and IV were all diagnosed with HIV following systemic illnesses that should be presumed to be due to immunocompromised state.
- One patient had esophageal candidiasis, four patients had recurrent viral infections, two had protozoal infections, two had intra cranial space occupying lesions (One expired during follow up, one diagnosed with tumefactive demyelination due to HIV infection), four patients had tuberculosis and the remaining seven patients had HIV wasting syndrome.
- Nine out of these twenty patients had HIV associated severe ocular manifestations. Interestingly one patient, a child of four years of age, in clinical stage IV (extra pulmonary TB) of age did not exhibit any ocular lesion and her CD4 count was 2149.
- The association of severe opportunistic ocular infection in WHO clinical Stage III/IV therefore has been statistically verified.

CD4 COUNT DISTRIBUTION: N = 100

- In our study we had CD4 count ranging from 28 – 2149 cells/mm³.
- The average CD4 count therefore was 503.57 cells /mm³.

Table 11

CD4 COUNT	NO OF PATIENTS
<200	11
201 – 500	45
>500	44

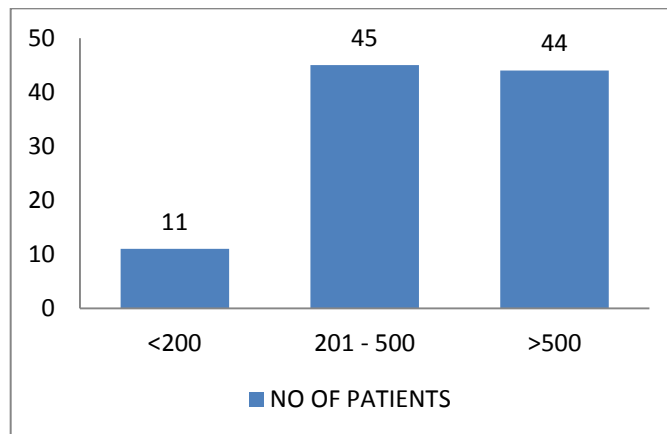


Table 12

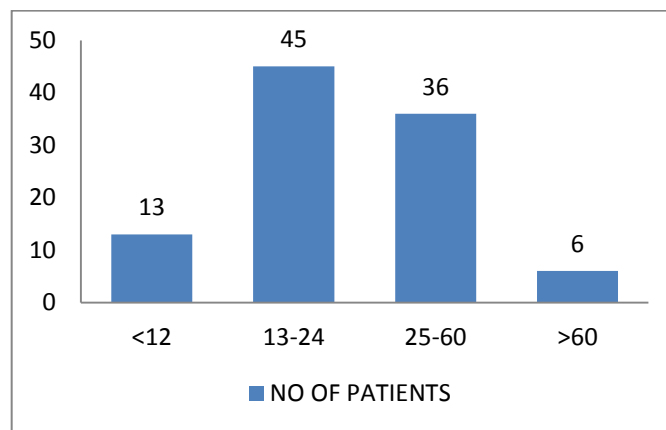
CD4 COUNT	AGE DISTRIBUTION				TOTAL
	<20	20-40	41-60	>60	
<200	0	7	3	1	11
200-500	3	29	12	1	45
>500	2	22	19	1	44
TOTAL	5	58	34	3	100

- Eleven patients were severely immunocompromised with CD4 count of < 200 cells/mm³.
- Note that none of the children observed in our study were severely immunocompromised. All five children maintained CD4 count > 200.

DURATION OF THE HIV DISEASE: N = 100

Table 13

DURATION SINCE DISEASE DETECTION (MONTHS)	NO OF PATIENTS
<12	13
13-24	45
25-60	36
>60	6
TOTAL	100

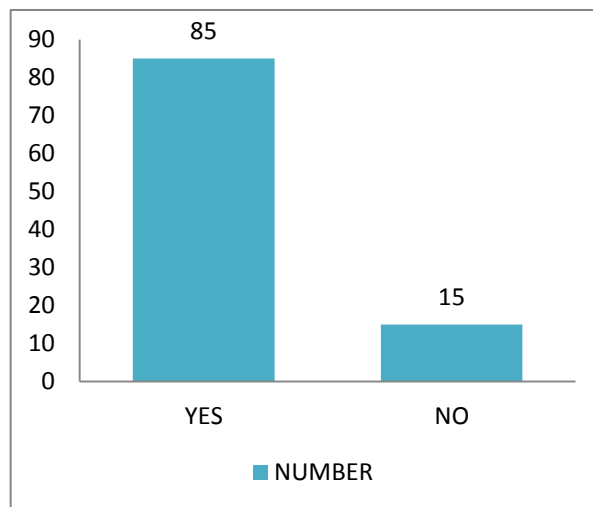


- We observed patients with disease duration ranging from as early as 2 months up to 9 years.
- The average duration of disease being 4.03 years.

HAART REGIME: N = 100

Table 14

PATIENTS ON HAART	NUMBER
YES	85
NO	15



- HIV seropositive patients detected with CD4 count of < 350 /mm³ at any point; at or later date to disease detection were started on HAART regime as per WHO recommendations.
- Fifteen patients were on observation with half yearly CD4 re-evaluation and treated symptomatically.
- Eighty-five patients were on HAART regime, which was initiated as per WHO recommendation, and as per patient's systemic associated conditions and response to drugs.

DURATION SINCE HAART TREATMENT: N = 85

Table 15

DURATION SINCE STARTING HAART (MONTHS)	NO OF PATIENTS
<12	15
13-24	17
25-60	47
>60	6
TOTAL	85

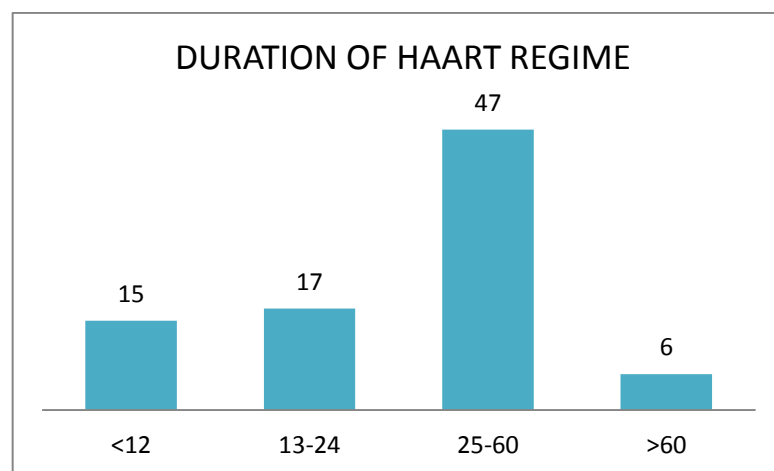


Table 16

DURATION SINCE STARTING HAART	OCULAR LESION		TOTAL
	PRESENT	ABSENT	
<12	12	3	15
13-24	12	5	17
25-60	38	9	47
>60	5	1	6
TOTAL	67	18	85
Chi Test = 0.827356 , Df =3			
P value= 0.2399			

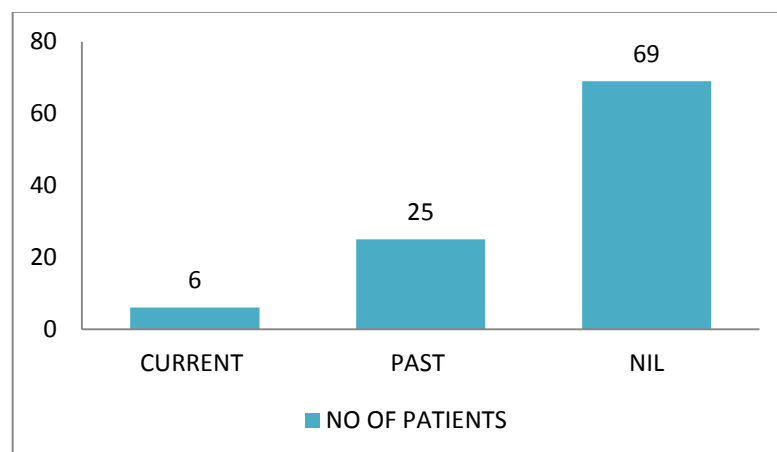
- Sixty-seven out of the eighty-five patients on HAART manifested ocular lesions in various forms.

- Majority of the lesion were found in time duration of two to five years of HAART regime.

TB STATUS: N = 100

Table 17

SYSTEMIC TB STATUS	NO OF PATIENTS
CURRENT	6
PAST	25
NIL	69
TOTAL	100



- Six out of hundred patients were recently diagnosed and currently under treatment for tuberculosis. Two of these were children of age less than fourteen.
- Twenty-five patients were treated for tuberculosis in the past, in the time frame of HIV detection- duration, of which one was a child.

OCULAR SYMPTOMS: N = 100

Table 18

OCULAR SYMPTOMS	NO OF PATIENTS
PRESENT	30
ABSENT	70

OCULAR FINDINGS: N = 100

Table 19

OCULAR FINDINGS	NO OF PATIENTS
PRESENT	78
ABSENT	22

- Seventy-eight out of the hundred patients that we examined had findings from the adnexa up to the optic nerve. The average CD4 count in these 78 patients was noted to be 468.16.
- These findings were either a manifestations of viral load, or of low immunity, and/ or of opportunistic infections.
- The twenty-two patients those who dint have ocular findings had an Average CD4 count of 629.09.

OCULAR SYMPTOMS VERSUS OCULAR FINDINGS:

Table 20

OCULAR FINDINGS	OCULAR SYMPTOMS		TOTAL
	PRESENT	ABSENT	
PRESENT	29	49	78
ABSENT	1	21	22
TOTAL	30	70	100
P value = 0.003178			

- Thirty HIV seropositive patients had ocular symptoms. Of these twenty-nine patients almost all had severe blinding opportunistic manifestations. The remaining one patient had refractive error.
- Amongst the remaining seventy patients who had no ocular symptoms HIV retinopathy was the most common finding.

VISION DISTRIBUTION:

The severity of manifestations on visual acuity was as observed below;

Table 21

VISION STATUS	RIGHT EYE	LEFT EYE	BOTH EYES
BLIND	3	2	1
<3/60	1	3	2
3/60- 6/60	1	0	0
6/60- 6/18	2	2	2
>6/18	93	94	87

- Five out of hundred patients had ocular manifestations leading on to bilateral impairment in vision. Of which one patient was completely blind.
- Nine out of hundred patients had unilateral vision impairing manifestations.
- The patient with bilateral blindness was diagnosed with severe toxoplasma retinochoroiditis.
- The remaining patients had manifestations like CMV retinitis, acute retinal necrosis, retinal vasculitis, retinitis, intermediate uveitis, viral keratitis, complicated cataract and non-healing fungal corneal ulcer.

OCULAR FINDINGS IN OUR STUDY:

- Seventy eight out of hundred patients had HIV related ocular manifestation. Majority of the patients in our study had multiple manifestations at the time of examination; blepharitis and conjunctival microvasculopathy were commonly found and accounting for the overlap in the total number of manifestations.

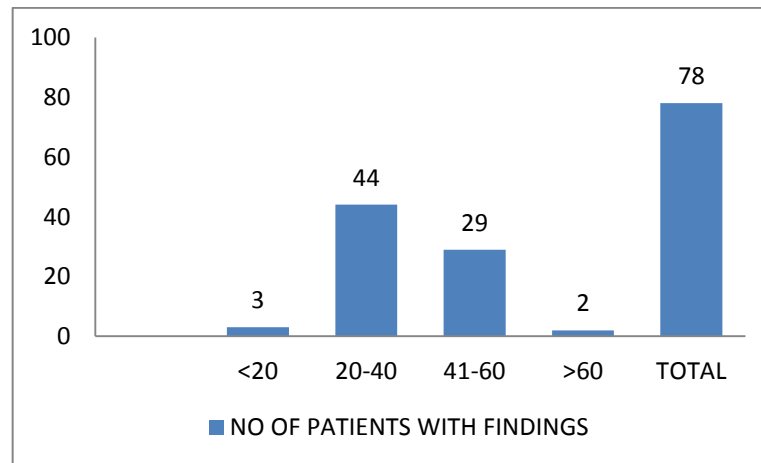
Table 22

OCULAR FINDINGS	NUMBER OF PATIENTS WITH FINDINGS	PERCENTAGE
ADNEXA		
BLEPHARITIS	29	24.79
MOLLUSCUM CONTAGIOSUM	2	1.71
STYE	2	1.71
HERPES ZOSTER	1	0.85
ALLERGIC / INFECTIVE CONJUNCTIVITIS	6	5.13
CONJ. MICROVASULOPATHY	53	45.3
ANTERIOR SEGMENT		
COMPLICATED CATARACT	1	0.85
HERPES ZOSTER OPHTHALMICUS	1	0.85
VIRAL KERATITIS	1	0.85
ANTERIOR UVEITIS	1	0.85
NON HEALING FUNGAL ULCER	1	0.85
INFLAMMATORY GLAUCOMA	1	0.85
POSTERIOR SEGMENT		
HIV RETINOPATHY	4	3.42
CMV RETINITIS BURNT OUT	2	1.71
ARN	1	0.85
TOXOPLASMOSIS	2	1.71
RETINAL VASCULITIS	1	0.85
INTERMEDIATE UVEITIS	1	0.85
RETINITIS	1	0.85
NEURO OPHTHAL		
MULTIPLE CRANIAL NERVE PALSY	1	0.85
OPTIC NEURITIS	1	0.85
OPTIC ATROPHY	4	3.42
ORBIT	NIL	

OCULAR FINDING AND AGE DISTRIBUTION: N = 78

Table 23

AGE IN YEARS	NO OF PATIENTS WITH FINDINGS
<20	3
20-40	44
41-60	29
>60	2
TOTAL	78

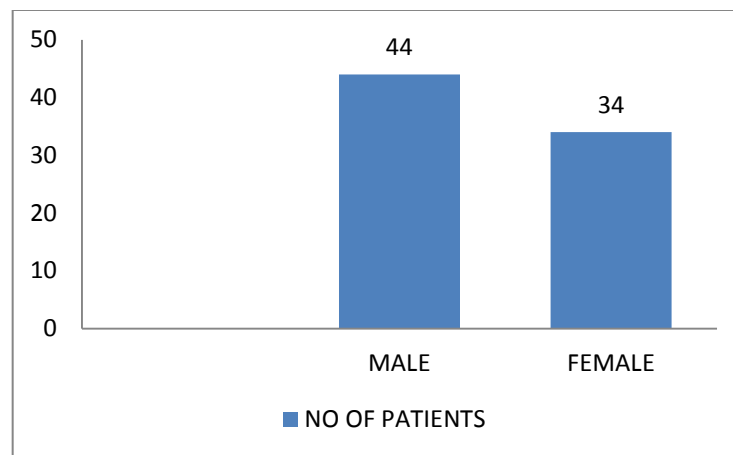


- 75.85 % of the ocular manifestation was seen in the age group of twenty to forty years (58 people).
- However when checked for its significance (by chi square test), the age wise distribution of ocular lesion proved to be statistically insignificant.

GENDER WISE DISTRIBUTION OF OCULAR FINDINGS: N = 78

Table 24

GENDER	NO OF PATIENTS	AVERAGE CD4 COUNT
MALE	44	432.32
FEMALE	34	606.09

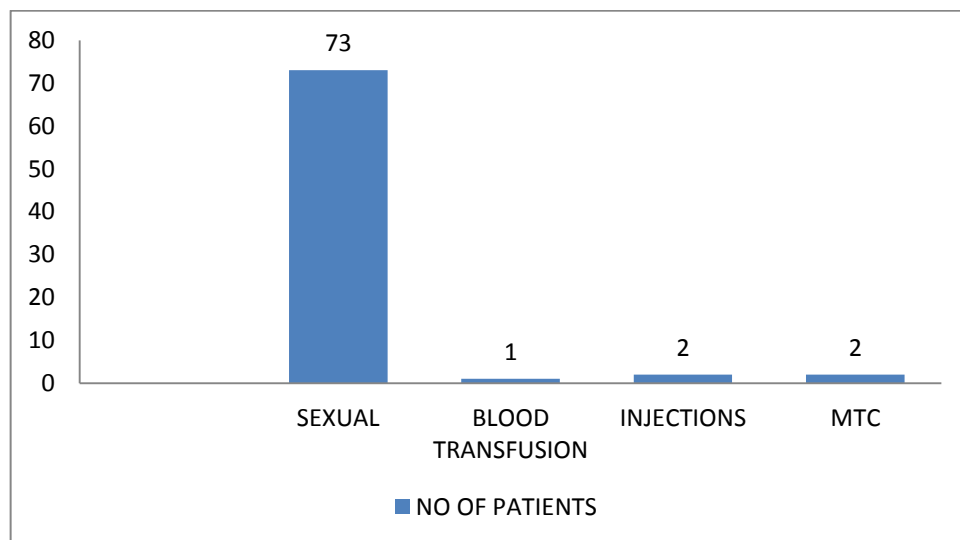


- Forty-four out of these seventy-eight patients with HIV ocular manifestations were male when comparison to thirty-four females; who had disease manifestations.
- The average CD4 count in the forty-four male patients with ocular manifestations was found to be 432.32.
- The average CD4 count of 606.09 was observed in thirty-four female patients with ocular manifestations.

OCULAR FINDING AND ROUTE OF EXPOSURE: N= 78

Table 25

MODE OF DISEASE EXPOSURE	NO OF PATIENTS	AVERAGE CD4 COUNT
SEXUAL	73	602.8
BLOOD TRANSFUSION	1	780
INJECTIONS	2	261
MTC	2	371.5



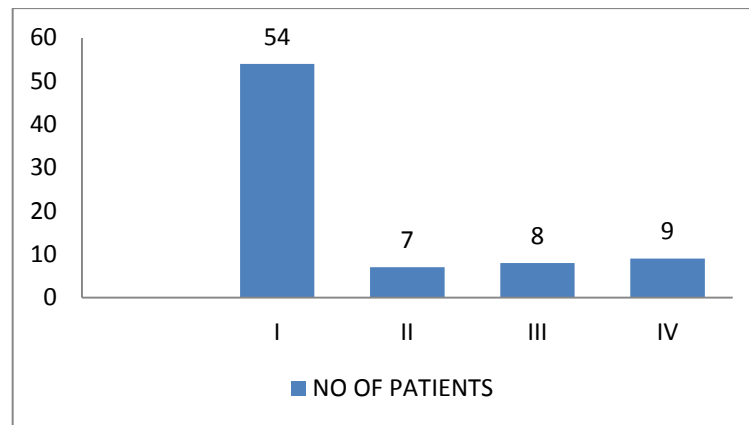
- This data shows the prevalence of manifestations for various routes of transmission.
- Injection/ IV drug abuse had 100% manifestations (though a non-blinding manifestation) when compared to blood transfusion which exhibited 50% prevalence of manifestation. However this data is insignificant in terms of sample size.

- Ocular manifestations were more seen in patients with sexual mode of transmission.
- Seventy-three of ninety-one (80%) were sexually exposed patients who exhibited ocular manifestations.
- Most of the ocular blinding manifestations were observed to occur in patients exposed to HIV sexually, and this specific group had CD4 ranging from 28- 1916 cell/mm³. And these in itself are manifestations of other associated sexually transmitted diseases.
- The 2 patients, who contracted disease by the IV route, had CD4 counts of < 350 cells/mm³. 1 out of the 2 patients, who had been seen after a year showed just a minimal improvement in immunity. This probably indicates the high viral load transmission via injections/IV drug abuse. However none of the two had opportunistic manifestations.
- Two of five children (40%) in our study group were observed to have manifested lesions.

OCULAR FINDINGS AND WHO CLINICAL STAGING: N = 78

Table 26

WHO CLINICAL STAGE	NO OF PATIENTS
I	54
II	7
III	8
IV	9
TOTAL	78



- Fifty-three out of the seventy-eight patients with ocular manifestation were observed under the clinical stage I of WHO classification. All patients in our study group with HIV retinopathy fell under this clinical staging of HIV. One patient with intermediate uveitis was under WHO clinical stage 1.
- Clinical stage II we observed seven people. Of these two patients had molluscum contagiosum, two had herpes zoster ophthalmicus, one had

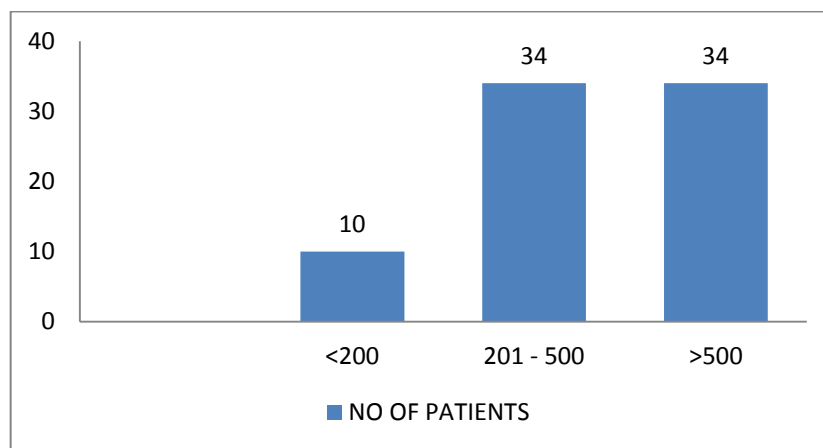
allergic conjunctivitis, one had non healing fungal corneal ulcer and one had microvasculopathy.

- Seventeen patients from WHO clinical stage III and IV categorised under ocular manifestations had vision impairing opportunistic diseases. These included viral keratitis, CMV retinitis , toxoplasma retinochoroiditis, acute retinal necrosis, multiple cranial nerve palsy, optic neuritis etc.
- The average CD4 count of patients from stage III and IV was 400.47 cells/mm³. The lowest CD4 count being 28 cells and the highest being 1043 cells/mm³.

OCULAR FINDING AND CD4 COUNT: N =78

Table 27

CD4 COUNT	OCULAR LESION		% OF OCULAR LESION
	PRESENT	ABSENT	
<200	10	1	90.90
200-500	34	11	75.55
>500	34	10	77.27



- The prevalence of ocular manifestations is noted to increase with decrease in CD4 count which can be observed from the table above.
- On the contrary, with the increase in CD4 count the prevalence of lesions were found to be decreasing which can be observed from the table above.

Table 28

CD4 COUNT	GENDER		TOTAL
	MALE	FEMALE	
<200	8	2	10
200-500	21	13	34
>500	14	20	34
TOTAL	43	35	78

- From the table above, we observed that the prevalence of male patient with ocular lesions is 80 % with the CD4 count < 200 cells/mm³.
- In the group with CD4 count > 500 cells/mm³, female and male patients had a prevalence of 58.82% and 41.17% respectively.

OCULAR FINDINGS AND HAART REGIME DURATION: N= 78

Table 29

DURATION OF HAART TREATMENT (MONTHS)	NUMBER OF OCULAR LESIONS	MEAN AND STD. DEV CD4 COUNTS				% INCREASE IN CD4 COUNT
		BASE CD4	STD DEV	PRESENT CD4	STD DEV.	
<12	12	315.33	194.64	314.66	217.31	-0.21
13-24	12	324.58	271.2	461.76	272.21	29.71
25-60	38	319.52	271.2	473.45	272.21	32.51
>60	5	425.16	270.2	766.16	273.04	44.51

Table 30

DURATION SINCE HAART TREATMENT	OCULAR LESION				TOTAL
	PRESENT	%	ABSENT	%	
<12	12	80	3	20	15
12-24	12	70.58	5	29.41	17
25-60	38	80.85	9	19.14	47
>60	5	83.33	1	16.66	6

- When comparing the baseline CD4 count at the start of HAART to the CD4 count at the time of examination, we observed a significant increase in CD4 count, suggesting the effectiveness of HAART.
- However we also observed an increase in ocular manifestations of HIV with increase in CD4 counts, while the patient was under HAART Regime.

CORRELATING THE FINDINGS WITH CD4 COUNTS: N =156 EYES

Table 31

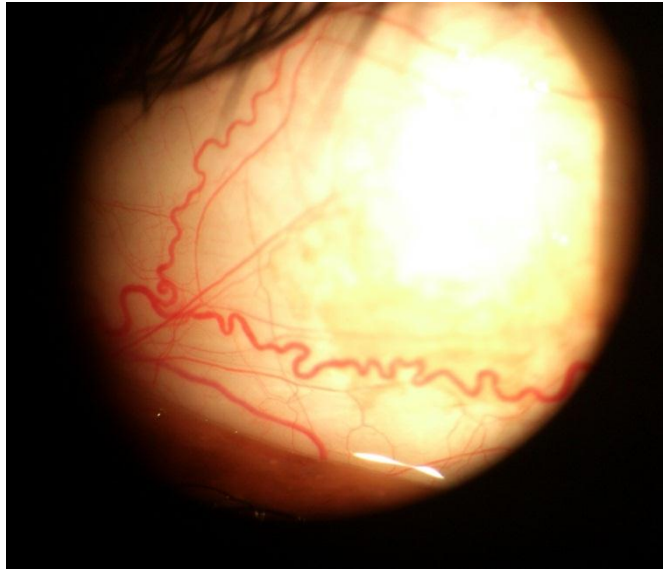
	CD4 COUNT DISTRIBUTION FOR N = 156		
ADNEXA	<200	200-500	>500
BLEPHARITIS	18	22	16
MOLLUSCUM CONTAGIOSUM	0	4	0
STYE	0	2	1
HERPES ZOSTER	0	1	0
ALLERGIC / INFECTIVE CONJUNCTIVITIS	2	8	2
CONJ. MICROVASCULOPATHY	19	34	52
ANTERIOR SEGMENT			
COMPLICATED CATARACT	0	2	2
HERPES ZOSTER OPHTHALMICUS	0	0	1
VIRAL KERATITIS	0	1	0
ANTERIOR UVEITIS	0	0	2
NON HEALING FUNGAL CORNEAL ULCER	0	1	0
INFLAMMATORY GLAUCOMA	0	0	2
POSTERIOR SEGMENT			
HIV RETINOPATHY	0	6	1
CMV RETINITIS BURNT OUT	0	0	3
ARN	2	0	0
TOXOPLASMOSIS	0	2	2
RETINAL VASCULITIS	0	0	2
INTERMEDIATE UVEITIS	0	0	2
RETINITIS	2	0	0
NEURO OPHTHAL			
MULTIPLE CRANIAL NERVE PALSY	1	0	0
OPTIC NEURITIS	1	0	0
OPTIC ATROPHY	2	0	6

From the above tabular column the following observations are made:

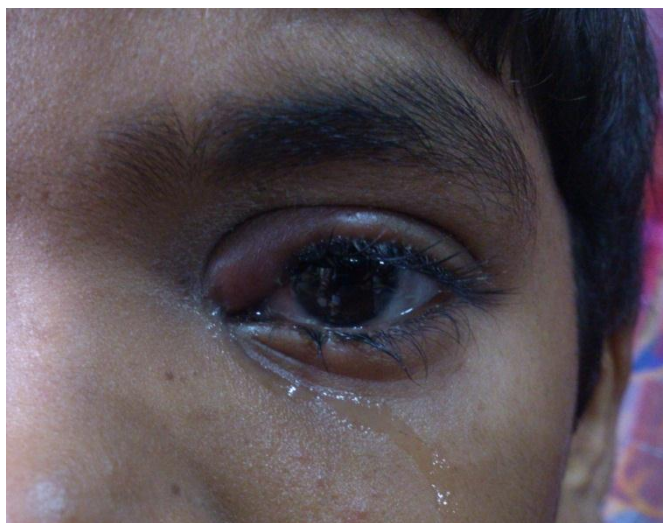
- Blepharitis was present in patients irrespective of the CD4 counts.

- Conjunctival microvasculopathy was present in patients irrespective of CD4 counts.

CONJUNCTIVAL MICROVASCULOPATHY



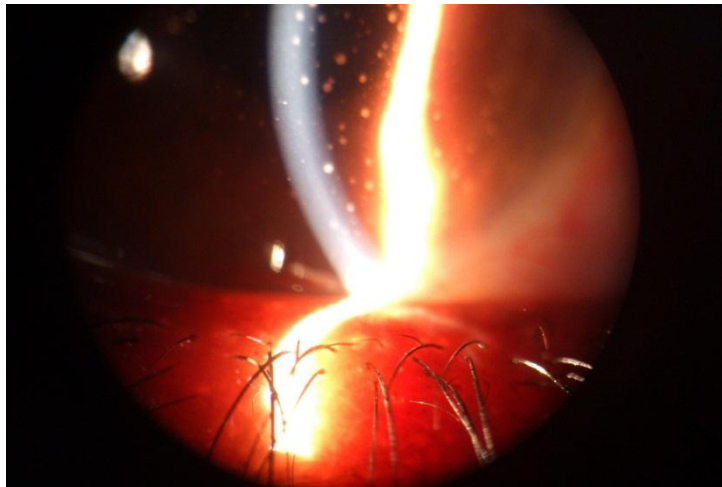
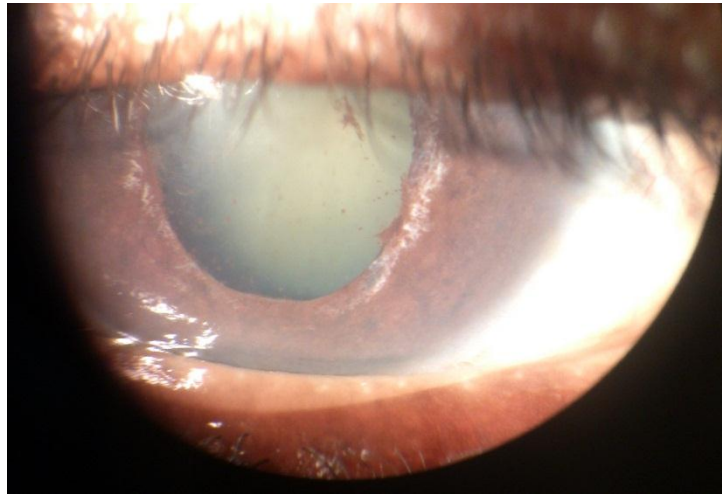
RECURRENT BLEPHARITIS



- Stye and molluscum contagiosum manifested in CD4 cell range of 200-500 cells.
- Allergic conjunctivitis was observed in all ranges of CD4 counts.
- Herpes zoster infection was seen to occur in CD4 of $> 200 - < 1000$ cells/mm³.
- Complicated cataract occurred respectively in patients with anterior uveitis and toxoplasmosis induced inflammatory anterior reaction in the respective CD4 cell ranges.
- Keratitis, both viral and fungal manifested in the range of 200 – 500 cells/mm³. Viral keratitis healed with patient's strict adherence to treatment.

The fungal ulcer was non-healing and referred to higher centre for surgical management.
- A special mention on HIV retinopathy : Out of four patients with HIV retinopathy, three manifestations occurred when CD4 had fallen < 500 cells/mm³. One was observed to have manifested with CD4 of > 500 cells/mm³.

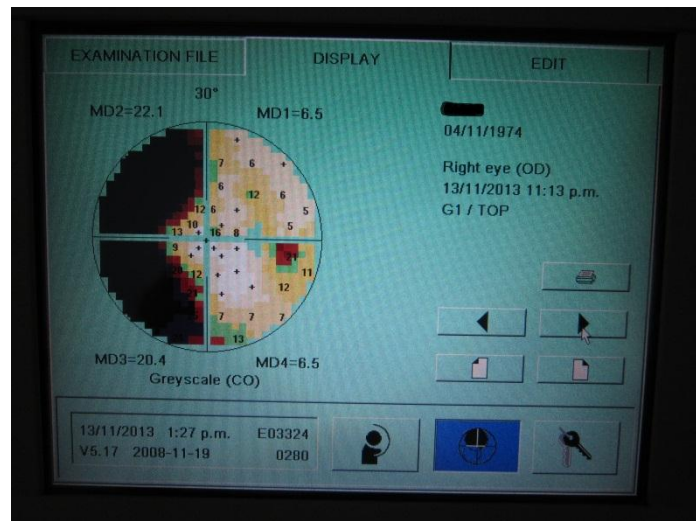
ANTERIOR UVEITIS WITH COMPLICATED CATARACT



HEALED HERPES ZOSTER



FIELD DEFECTS IN PATIENT WITH TUMEFACTIVE DEMYELINATION OF BRAIN



- It should also be noted that these patients with HIV retinopathy had associated conjunctival microvasculopathy.

- The two patients we examined to have CMV retinitis, both females; had burnt out retina. And both these patients had their CD4 > 500 cells.

This indicated a probable infection with CMV when severely immunocompromised, and healing of lesion with adequate treatment.

- One patient presented to us with Acute Retinal Necrosis with a CD4 count of 28cells/mm³. The patient expired during follow up.

- The intermediate uveitis was of unknown etiology. All necessary investigation for TORCH and PCR for herpes and varicella came negative. Patient was not on any uveitis inducing drugs.

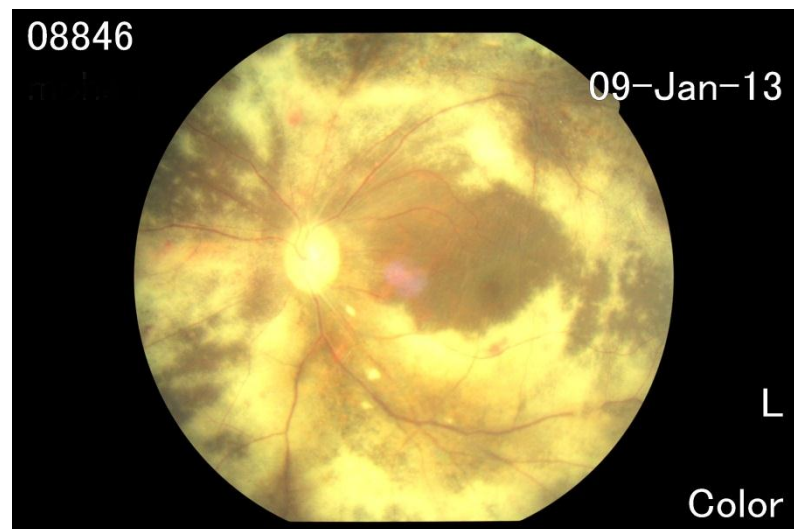
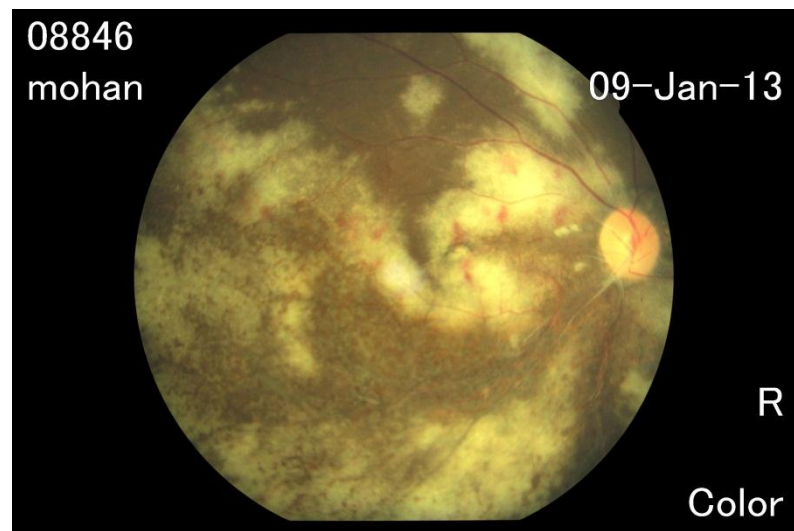
This probably was a case of HIV induced uveitis.

- The patient with features of retinal vasculitis and no systemic manifestation of tuberculosis (CD4 of 1043 cells/mm³) failed to review with reports and therefore the laboratory diagnosis was incomplete.

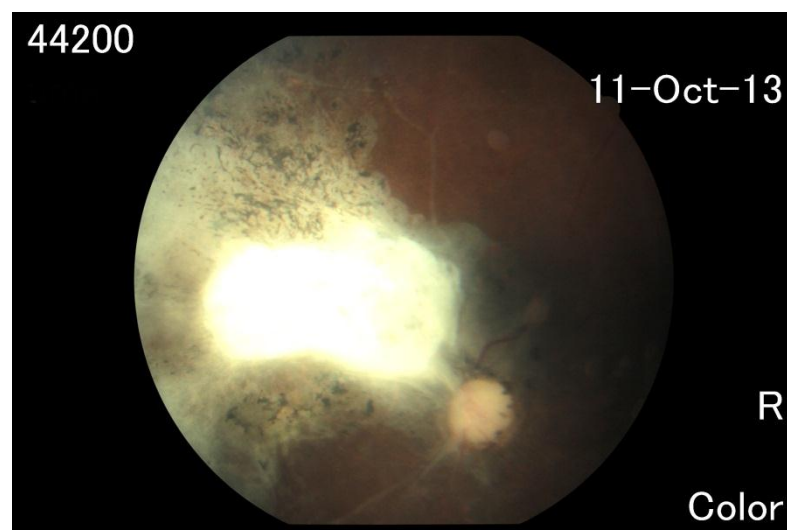
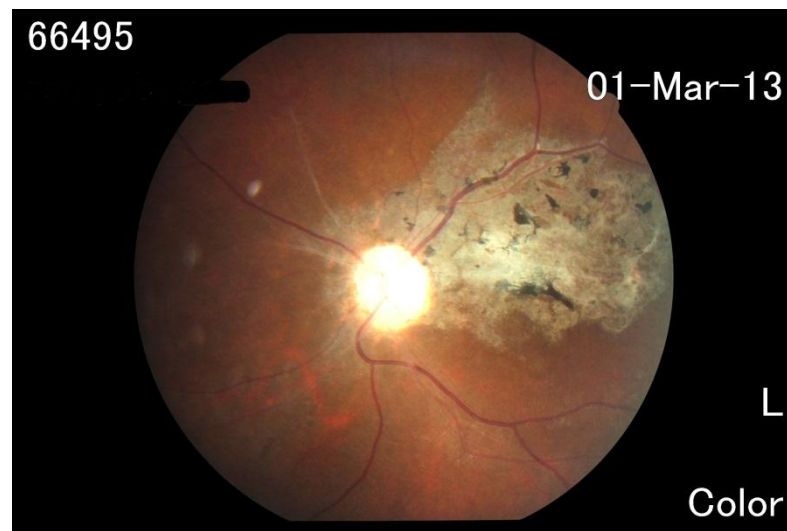
- Retinitis was seen in one patient involving both eyes with CD4 count of < 200 cells / mm³. Patient was positive for toxoplasma gondii, herpes simplex virus and cytomegalovirus.

- Multiple cranial nerve palsy was observed in one patient, who had multiple space occupying lesions in the brain on follow up CT brain.

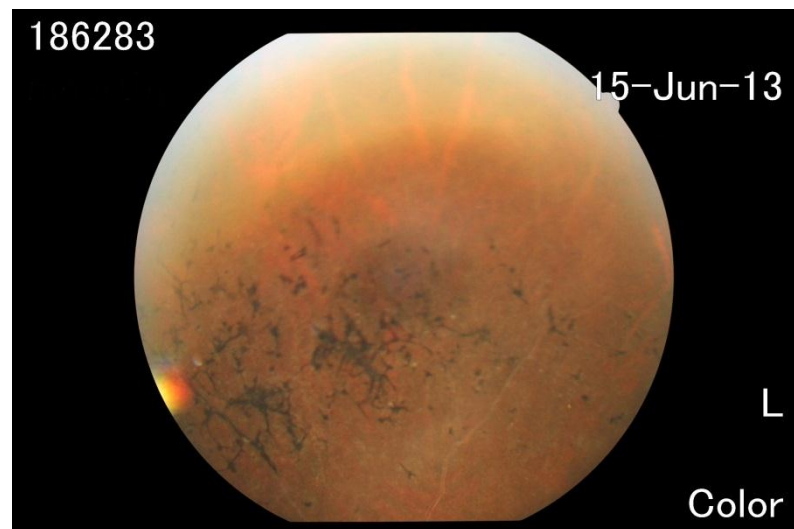
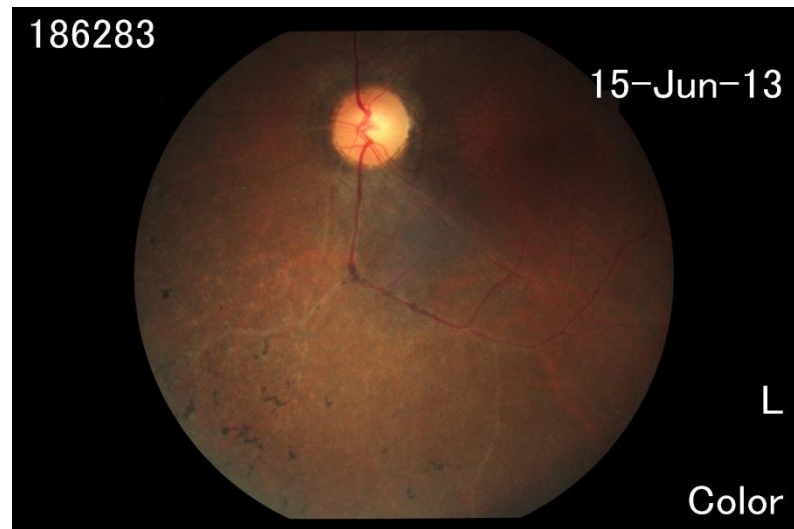
ACUTE RETINAL NECROSIS



CYTOMEGALOVIRUS RETINITIS – BURNT OUT RETINA



UN SPECIFIED RETINITIS IN HIV

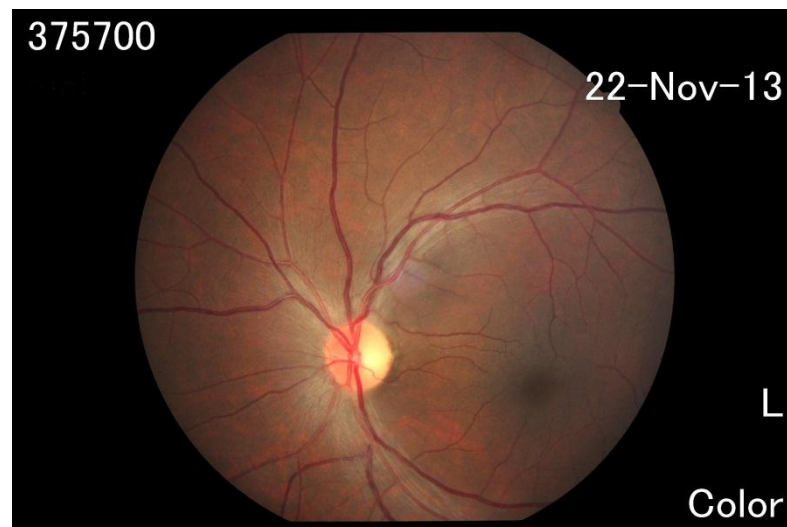
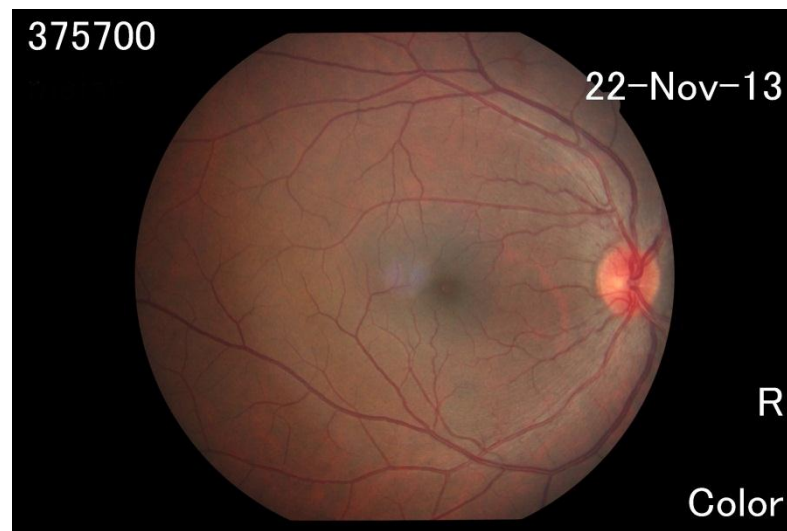


- However the exact nature of the lesions remained unknown as the patient expired during follow up.
- One patient presented himself with atypical optic neuritis associated with hemiparesis and fever. Thorough systemic evaluation revealed multiple space occupying lesions in the brain. Patient's CD4 count was found to be 100 cells/ mm. After battery of tests it was diagnosed to be HIV white matter disease, progressive in nature.

The exact diagnosis was Tumefactive demyelination disease.

- Consecutive optic atrophy was observed in three patients with retinal changes. One patient had HIV associated primary optic atrophy.

OPTIC NEURITIS AND OPTIC DISC PALLOR



DISCUSSION

SOCIODEMOGRAPHIC STATISTICS

AGE WISE STATISTICS:

Table 32

AGE	BISWAS ET AL	GURURAJ ET AL	LAMICHHANE G ET AL	PRESENT STUDY
N	100	100	117	100
<20	7%	7%	NA	5%
20 -40	74.60%	54%	78.60%	58%
41-60	19%	36%	NA	34%
>60		3%	NA	3%
MEAN AGE	34.16	36.4	30.14+/- 11.82	38.14+/- 11.84

- The mean age of patients in our study was 38.14 +/- 11.84 which fell in line with mean age of the comparative studies.
- Majority of the patient prevalence in our study fell under the reproductive age group of 20-40 years. This pattern was similarly noticed in Biswas et al , Gururaj et al and Lamichhane G et al study. However our prevalence rate of 58 % was closely comparable to the Gururaj et al study which had 54 %. Also comparable with the Gururaj et al study was the decrease in prevalence with increase in age.
- Of special mention is the similarity in the prevalence of patients in the age group < 20 years.

GENDER STATISTICS:

Table 33

GENDER	BISWAS ET AL	GURURAJ ET AL	LAMICHHANE G ET AL	AMARE ET AL	PRESENT STUDY
N	70	100	117	126	100
MALE	74%	46%	64.90%	34.12%	59%
FEMALE	21.42%	54%	35.10%	65.87%	41%

- Our study shows a higher male prevalence in comparison to females. This finding was similarly noted in Biswas et al and Lamichhane G et al study.
- As previously mentioned the age gender distribution in our study was not significant, $p= 0.3$. This was also the finding in Lamichhane G et al study.

MODE OF TRANSMISSION DISTRIBUTION:

Table 34

MODE OF TRANSMISSION	BISWAS ET AL	GURURAJ ET AL	PRESENT STUDY
	N=70	N=100	N=100
SEXUAL	60%	81%	91%
BLOOD TRANSFUSION	18.50%	1%	2%
INJECTIONS	7.10%	NA	2%
MTC	7.10%	7%	5%
			P= 0.1274

- Sexual route was the most common mode of disease transmission, and this was observed in study by Biswas et al and Gururaj et al. Gururaj et al had 1 homosexual route of exposure.
- In our study heterosexual route was the only sexual route of transmission and this finding was consistent with the Biswas et al study.
- All 5 children in our study acquired infection from vertical transmission.
- Two males from reproductive age acquired infection by sharing of needle.
- P value of 0.1274 signifies that the route of exposure has no impact the prevalence of the ocular manifestations.

WHO CLINICAL PROFILE:

Table 35

WHO CLINICAL STAGING	GURURAJ ET AL	AMARE ET AL	PRESENT STUDY
N	100	126	100
I	62%	63.50%	73%
II		13%	7%
II	38%	17.50%	10%
IV		6.35%	10%
P	<0.05	<0.01	0.003

- Majority of the patients in our study were observed in clinical stage I and II. The findings were similar in the Gururaj et al and Amare et al study.
- It was observed in our study that all patients those who fell under the Clinical category III /IV had ocular HIV manifestation, and mostly of the opportunistic type. This was equally observed in the studies to which we compared to in the above table.
- With p value of 0.003 we can conclude that prevalence of ocular manifestation increases with the clinical staging of disease. And this is probably due to the increase in the opportunistic infection in these stages. This was observed both in Gururaj et al and the Amare et al study as well.

CD4 COUNT AND EYE INVOLVEMENT:

Table 36

CD4 COUNTS	GURURAJ ET AL	PATHAI ET AL	AMARE ET AL	PRESENT STUDY
N	46	26	27	78
<200	92.90%	76.92%	25.90%	15%
200-500	7.10%	23.10%	48.10%	50%
>500			25.90%	34%
P	0.001	<0.01	<0.01	0.0235

- We observed an increased prevalence of ocular manifestations in the CD4 range of 200 – 500 cells/mm³. This was comparable to the Ethiopian study by Amare et al.
- However the prevalence of ocular manifestations in the range < 200 cells/mm³ fell drastically in comparison Gururaj et al and Pathai et al, and significantly with Amare et al.
- P value in our study was 0.0235, which was significant. This significance was observed in all the above mentioned studies.
- Hence ocular manifestations is seen to increase significantly with decrease in CD4 count, especially in levels < 200 cells/mm³.

OCULAR MANIFESTATIONS

ANTERIOR SEGMENT FINDINGS:

Table 37

OCULAR FINDINGS	GURURAJ ET AL (DAVANGER E 2013)	LAMICHHA NE G ET AL (NEPAL 2010)	AMARE ET AL(NW ETHIOPIA 2011)	BEKELE ET AL (ETHIOPIA 2013)	PRESENT STUDY (2013)
N	100(%)	117(%)	126(%)	348(%)	100(%)
BLEPHARITIS	1	4(2.5)	3(2.38)	11(3.2)	29
MOLLUSCUM CONTAGIOSUM	1	0	3(2.38)	9(2.6)	2
STYE	3	0	0	0	2
HERPES ZOSTER	42	4(4.27)	3(2.38)	3(0.8)	2
KAPOSI SARCOMA OF ADNEXA	0	0	1(0.7)	2(0.5)	0
CONJUNCTIVAL SCC/ LYMPHOMA	0	0	1(0.7)	8(2.3)	0
SCLERITIS	1	0	0	0	0
CONJUNCTIVITIS	0	3(1.7)	0	0	6
CONJUNCTIVAL MICROVASCULOPA THY	1	0	0	8(2.3)	78
DRY EYE	3	0	1(0.7)	40(11.5)	0
KERATITIS(BACTE RIAL/VIRAL/FUNG AL)	34	0	1(0.7)	2(0.5)	2
ANTERIOR UVEITIS	21	0	5(3.9)	1(0.2)	2
COMPLICATED CATARACT	NA	0	0	0	2
INFLAMMATORY GLAUCOMA	NA	0	0	0	1

- Blepharitis and conjunctival microvasculopathy was the commonest anterior segment findings observed in this study. The prevalence of both of which were significantly higher in comparison to the studies mentioned above.
- Kaposi sarcoma and conjunctival squamous cell carcinoma were nil, and were comparable to the two Indian studies mentioned above. These

findings were present in the African studies. And this has been implicated due to the homosexual practises.

- Comparing to the two Indian studies, we see differences in most of the anterior segment presentations. This can be attributed to the geographical change in location of the study.

Table 37

OCULAR FINDINGS (N=100)	BISWAS ET AL (2008)	PRESENT STUDY
N	100	100
BLEPHARITIS	1	28
MOLLUSCUM	2	2
HERPES ZOSTER OPHTHALMICUS	6	2
CONJUNCTIVAL MICROVASCULOPATHY	NA	78
ANTERIOR UVEITIS	4	2
KAPOSI SARCOMA	1	0

- On comparing our study with Biswas et al, we found a similar prevalence of blepharitis and conjunctival microvasculopathy. Biswas et al mentions in text both the lesion to be the most common anterior segment manifestation. The one important patient mentioned above was of non-healing staphylococcal blepharitis.
- One patient with Kaposi sarcoma was from Dubai.

POSTERIOR SEGMENT:

Table 38

OCULAR FINDINGS	BISWAS ET AL (CHENNAI 2013)	GURURAJ ET AL (DAVANGERE 2013)	LAMICHHANEG ET AL (NEPAL 2010)	AMARE ET AL (NORTHWEST ETHIOPIA 2011)	BEKELE ET AL (ETHIOPIA 2013)	PRESENT STUDY (2013)
N	1000(%)	100	117(%)	126	348	100
HIV RETINOPATHY	43(4.3)	12	19(16.23)	9	2(0.6)	4
CMV RETINITIS	248(24.8)	7	5(4.23)	0	0	2
TOXOPLASMOSIS	28(2.8)	1	2(1.7)	7	1(0.3)	2
ACUTE RETINAL NECROSIS	11(1.1)	1	0	12	0	1
INTERMEDIATE UVEITIS	18(1.8)	0	0	0	0	1
OCULAR TB	26(2.6)	4	1(0.9)	0	0	0
OCULAR SYPHILIS	0	0	0	0	0	0
OTHER RETINAL DISEASES	NA	3	3(2.6)	0	1	2
CRVO	3(0.3)	0	0	0	0	0
ENDOPHTHALMITIS	7(0.7)	0	0	0	0	0

- The prevalence of HIV retinopathy, toxoplasmosis acute retinal necrosis and uveitis in our study was comparable to the prevalence in Biswas et al study.

The same finding was not so with CMV retinitis. This limitation was observed probably due to the sample size in our study.

- Ocular TB and syphilis were not recorded in our study.

NEURO-OPHTHAL MANIFESTATIONS:

Table 39

OCULAR FINDINGS	BISWAS ET AL (CHENNAI 2013)	GURURAJ ET AL (DAVANGERE 2013)	LAMICHHANEG ET AL (NEPAL 2010)	AMARE ET AL (NW ETHIOPIA 2011)	BEKELE ET AL (ETHIOPIA 2013)	PRESENT STUDY (2013)
N	1000	100	117(%)	126(%)	348(%)	100
CRANIAL NERVE PALSY	5(0.5)	4	1	1	5(1.5)	6
PAPILLEDEMA	0	1	2	0	0	0
OPTIC NEURITIS	0	0	1	0	4(1.2)	1
OPTIC ATROPHY	19 (1.9)	0	0	0		4

- From the above table we observe that cranial nerve palsy was the most common neuro ophthalmological manifestation of HIV. Followed by optic neuritis and optic atrophy.

ORBIT:

Table 40

OCULAR FINDING	BISWAS ET AL (2013)	GURURAJ ET AL (DAVANGERE 2013)	LAMICHHANEG ET AL (NEPAL 2010)	AMARE ET AL (NW ETHIOPIA 2011)	BEKELE ET AL (ETHIOPIA 2013)	PRESENT STUDY (2013)
OBITAL CELLULITIS	1	1	0	0	0	0
ORBITAL TUMOR	0	0	0	0	0	0

- We observed no case of orbital cellulitis or any orbital manifestations of HIV and this prevalence rate was comparable other studies.

CONCLUSION

- HIV infection is a problematic communicable disease present in our population, affecting commonly the reproductive age group.
- Disease awareness and prevention programme must be undertaken targeting most importantly the unskilled segment of population.
- A proper screening of blood units in the blood banks is a must to prevent risk of undue HIV transmission to patients.
- Screening of antenatal women with ELISA and taking necessary steps to prevent mother to child transmission will minimise the risk of the disease in children.
- HIV manifests in the eye either directly in the form of viral load or causes low immunity thereby increasing chances for opportunistic infections.
- With the introduction of HAART, the life expectancy of the patients have significantly increased. However the ocular manifestations continue to present in innumerable forms.

- Not all patients with early HIV opportunistic infection present to us with ocular symptoms until unless the manifestation is severely blinding and irreversible.
- Most of the symptomatic patients are the ones with blepharitis and conjunctivitis, a non-blinding yet troublesome form of disease manifestations.
- The ART centres in India at present practice just the referral of patients for ophthalmological examination only when the patient develops ocular complaints.
- With the number of ocular findings observed, our study highlights the need for a routine ophthalmological screening of all HIV seropositive patients.
- We recommend a routine screening of HIV seropositive patients upon diagnosis, prior to starting ART therapy to obtain a baseline ocular status.

- Once the patient is started on HAART , he/she must undergo at-least a half yearly ocular examination. This is important for two reasons; one to look out for immune reconstitution syndromes, two to identify the ocular side effects of HAART.

- CD4 counts have to be strictly considered while monitoring these patients. It serves both as a risk factor as well as an indicator of opportunistic manifestations.

- A very important observation we made was the patients non-consenting to any detailed examination outside of the ART centre. This was due to the stigma attached to the disease and the fear of being publically recognised as HIV seropositive.

- All this indicates a need for provision of ophthalmic setup in the ART centre.

- This can be made possible with adequate resources and trained ophthalmic personnel.

ANNEXURES

ABBREVIATIONS

HIV	HUMAN IMMUNODEFICIENCY VIRUS
AIDS	ACQUIRED IMMUNODEFECIENCY SYNDROME
CD	CLUSTER OF DIFFERENTIATION
RNA	RIBONUCLEIC ACID
WHO	WORLD HEALTH ORGANISATION
CDC	CENTRE FOR DISEASE CONTROL
ART	ANTIRETROVIRAL THERAPY
HAART	HIGHLY ACTIVE ANTIRETROVIRAL THERAPY
ELISA	ENZYME LINKED IMMUNOSORBENT ASSAY
CMV	CYTOMEGALOVIRUS
ARN	ACUTE RETINAL NECROSIS
MTC	MOTHER TO CHILD TRANSMISSION

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PROFORMA

Name

Age

Sex

Occupation

Marital status

Pre ART/ ART no.

Mode of transmission

When diagnosed

Who clinical stage

Duration since diagnosis

CD4 count baseline

CD4 count at present

Haart initiated

Drug administered

Associated systemic illness if any

Ocular complaints /symptoms and its duration:

OCULAR EXAMINATION

Preliminary examination:

	RE	LE
VISION		
EYELID/LASH		
CONJUNCTIVA		
CORNEA		
ANTERIOR CHAMBER		
IRIS		
PUPIL		
LENS		
IOP		
SCHIRMERS		

SLIT LAMP EXAMINATION AND 90D

RIGHT EYE:

LEFT EYE:

DILATED FUNDUS EXAMINATION WITH IDO

Right eye:

left eye:

DIAGNOSIS:

TREATMENT:

தகவல் படிவம்

தலைப்பு: ஹச்.ஐ.வி.நோயின் கண்கள் சார்ந்த அறிகுறிகளையும் மேலும் அதனை CD-4 எண்ணிக்கையுடன் ஒப்பிடும் ஓர் ஆய்வு

பங்கு பெருபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

மேலே குறிப்பிட்ட ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான விலக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

இதில் எனது கண்களை பரிசோதித்து, அதில் HIV யின் அறிகுறிகளை ஆராய்ந்து நான் முழு மனதுடன் தன்னிச்சையாக சம்மதிக்கிறேன்.

எந்த காரணத்தினாலோ, எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வய்வில் இருந்து விலகி கொள்ளலாம் என்று அறிந்து கொண்டேன்.

இதில் மருத்துவர் என் மீது எந்த ஊசியோ, பங்கேற்பவர்கள் பாதிக்கப்படும் பரிசோதனையோ (Invasive diagnostic test) செய்யப் போவதில்லை என அறிந்து கொண்டேன்.

இந்த ஆய்வு சமந்தமாகவோ இதைச் சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும், இவ்வய்வில் பங்குபெறும் மருத்துவர் என் மருத்துவ அறிக்கைகளை பார்க்க என் அனுமதி தேவையில்லை. இதன் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் பார்ப்பதற்கும், பயன்படுத்துவதற்கும் முழு மனதுடன் சம்மதிக்கிறேன்.

எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வேன் எதிர்பாராத வழக்கத்திற்கு மாறான நோய் அறிகுறி தென்பட்டால் அதை மருத்துவரிடம் தெரிவிப்பேன்.

இந்த ஆய்வில் நான் தன்னிச்சையாக தான் பங்கேற்கிறேன் எந்த காரணத்தினாலோ, எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வய்வில் இருந்து விலகி கொள்ளலாம் என்று அறிந்து கொண்டேன்.

இந்த ஆய்வில் எனக்கு தேவையான அனைத்து பரிசோதனையும் செய்து கொள்ள முழு மனதுடன் சம்மதிக்கிறேன். மேலும் இந்த ஆய்வில் என்னையும், என் கண்ணில் உள்ள பிரச்சினைகளையும் புகைப்படம் எடுக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்/
கட்டை விரல் ரேகை

நாள்

நான், இவ்வாய்வைப் பற்றி அனைத்து விபரங்களையும் மேற்குறிப்பிட்ட நபர் புரிந்து கொள்ளும்படி அவருக்கு தெரிந்த மொழியில் எடுத்துக்கூறி சம்மதம் பெற்றுள்ளேன்.

ஆய்வாளரின் பெயர் மற்றும் கையொப்பம்

நாள் :

SL NO	NAME	PRE ART/ ART NO	AGE	SEX	RELIGION	MARITAL STATUS	OCCUPAT ION	MODE OF TRANSMISS ION	YEAR OF HIV DETECTION	BASELINE CD4	DATE OF EXAMIN ATION	PRESEN T CD4	DURATION SINCE DETECTIO N	WHO STAGE OF DISEA SE	HAAR T REGI MEN	DURATIO N SINCE TREATME NT	OCULAR SYMPTO MS	TB STATUS	BEST CORRECTED		RIGHT EYE		LEFT EYE		ADNEXA		ANTERIOR SEGMENT	
																			VISUAL ACUITY									
																			RIGHT	LEFT								
1	M	200/201 1	25	M	HINDU	N	COOLIE	SEXUAL	2013	28	2013	28	< 12 MONTHS	3	Y	< 12 MONTHS	YES	NIL	PL +	PL+	BLEPHARIT IS	CONJ. MICROVASCULOPATH Y						
2	RB	82/2006	30	F	MUSLIM	W	HOUSEWI FE	SEXUAL	2006	870	2013	920	> 60 MONTHS	4	Y	> 60 MONTHS	YES	NIL	6/6	PL+	-	-						
3	SB	67/2006	7	F	MUSLIM	N	SCHOOL	MTC	2006	140	2013	1007	> 60 MONTHS	1	Y	> 60 MONTHS	NO	PAST	6/6	6/6								
4	U	345/201 1	35	F	HINDU	Y	HOUSEWI FE	SEXUAL	2011	56	2013	504	25 - 60 MONTHS	4	Y	25- 60 MONTHS	YES	NIL	PL -	6/6	-	-						
5	P	907/201 2	4	F	HINDU	N	SCHOOL	MTC	2012	2149	2013	2149	13 - 24 MONTHS	4	N	NO	NO	CURREN T	6/6	6/6								
6	M	924/201 2	25	F	HINDU	Y	HOUSEWI FE	SEXUAL	2012	336	2013	336	13 - 24 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATH Y						
7	S	935/201 2	29	M	HINDU	Y	CLERK	SEXUAL	2012	397	2013	397	13 - 24 MONTHS	1	N	NO	NO	NIL	6/6	6/6								
8	A	677/201 2	32	M	HINDU	Y	ATTENDE R	SEXUAL	2012	150	2013	169	13 - 24 MONTHS	4	Y	13- 24 MONTHS	YES	CURREN T	6/24	6/24	BLEPHARIT IS	CONJ. MICROVASCULOPATH Y						
9	S	101/201 0	57	F	HINDU	Y	HOUSEWI FE	SEXUAL	2010	129	2013	745	25 - 60 MONTHS	3	Y	25- 60 MONTHS	YES	PAST	PL-	PL+	BLEPHARIT IS	CONJ. MICROVASCULOPATH Y ,INFLAMMATORY GLAUCOMA						
10	D	186/200 9	63	M	HINDU	Y	PAINTER	SEXUAL	2009	297	2013	496	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/18	6/24	-	CONJ. MICROVASCULOPATH Y						
11	S.M	326/201 1	32	F	HINDU	Y	HOUSEWI FE	SEXUAL	2011	32	2013	445	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATH Y						
12	R	702/201 2	46	M	HINDU	Y	DRIVER	SEXUAL	2012	320	2013	219	13 - 24 MONTHS	1	Y	13- 24 MONTHS	YES	PAST	6/6	6/6	-	ALLERGIC CONJUNCTIVITIS, CONJ MICROVASCULOPATH Y						
13	N	579/200 9	48	M	HINDU	Y	DAILY WAGER	SEXUAL	2009	280	2013	350	25 - 60 MONTHS	2	Y	25- 60 MONTHS	YES	PAST	6/6	HM	BLEPHARIT IS	-						

14	U	9621/2011	28	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	688	2013	650	25 - 60 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
15	PD	566/2010	34	M	HINDU	Y	COOLIE	SEXUAL	2010	361	2013	459	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
16	D	3013/2013	38	M	HINDU	Y	COOLIE	SEXUAL	2013	194	2013	194	< 12 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6		
17	S	417/2013	26	M	HINDU	Y	HOTEL WORKER	SEXUAL	2009	690	2013	348	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
18	M	335/2008	41	M	HINDU	Y	COOLIE	SEXUAL	2008	72	2013	150	25 - 60 MONTHS	4	Y	25- 60 MONTHS	YES	PAST	TUNNEL VISION	TUNNEL VISION	BLEPHARITIS	CONJ. MICROVASCULOPATHY
19	S	1111/2012	24	M	HINDU	Y	DRIVER	SEXUAL	2012	812	2013	467	13 - 24 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6		
20	M	318/2011	36	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	240	2013	775	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
21	D	1235/2012	28	F	HINDU	Y	HOUSEWIFE	SEXUAL	2012	501	2013	540	13 - 24 MONTHS	1	N	NO	NO	NIL	6/6	6/6		
22	R	370/2010	45	M	HINDU	Y	SELF EMPLOYED	SEXUAL	2010	402	2013	593	25 - 60 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
23	M	229/2010	55	F	HINDU	Y	HOUSEWIFE	SEXUAL	2009	171	2013	148	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/12	6/12	-	CONJ. MICROVASCULOPATHY
24	V	375/2011	48	M	HINDU	Y	DRIVER	SEXUAL	2010	249	2013	647	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY
25	S	443/2011	46	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	262	2013	646	25 - 60 MONTHS	1	Y	25- 60 MONTHS	YES	NIL	6/6	6/6	BLEPHARITIS	ALLERGIC CONJUNCTIVITIS
26	P	285/2010	77	M	HINDU	W	UNEMPLOYED	SEXUAL	2010	86	2013	113	25 - 60 MONTHS	3	Y	25- 60 MONTHS	NO	PAST	6/60	6/60	BLEPHARITIS	-
27	I	675/2012	45	F	HINDU	Y	COOLIE	SEXUAL	2012	265	2013	651	13 - 24 MONTHS	3	Y	13- 24 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY
28	C	806/2013	34	M	HINDU	Y	DRIVER	SEXUAL	2013	235	2013	300	< 12 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6	BLEPHARITIS	-

29	A	605/2012	34	F	HINDU	Y	DAILY WAGER	SEXUAL	2012	193	2013	210	13 - 24 MONTHS	1	Y	13- 24 MONTHS	YES	PAST	6/6	6/6	-	ALLERGIC CONJUNCTIVITIS, CONJ. MICROVASCULOPATHY
30	E	429/2011	35	M	HINDU	Y	LOADMAN	SEXUAL	2011	349	2013	597	25 - 60 MONTHS	2	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
31	G	233/2010	36	F	HINDU	Y	HOUSEWIFE	SEXUAL	2010	241	2013	243	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
32	R	898/2011	20	M	HINDU	N	STUDENT	IV DRUG ABUSE	2013	210	2013	210	< 12 MONTHS	2	Y	< 12 MONTHS	YES	NIL	6/6	6/6	MOLLUSCUM CONTAGIOSUM	-
33	P	534/2012	35	M	HINDU	Y	UNEMPLOYED	SEXUAL	2010	441	2013	307	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	CURRENT	6/6	6/6		
34	G	665/2012	13	M	HINDU	N	SCHOOL	MTC	2012	194	2013	331	13 - 24 MONTHS	3	Y	13- 24 MONTHS	NO	CURRENT	6/6	6/6		
35	E	630/2012	67	M	HINDU	Y	UNEMPLOYED	SEXUAL	2012	431	2013	571	13 - 24 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/36	6/36		
36	S	732/2011	57	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	253	2013	312	25 - 60 MONTHS	1	Y	25- 60 MONTHS	YES	NIL	6/12	6/12	BLEPHARITIS	CONJ. MICROVASCULOPATHY
37	P	282/2010	32	M	HINDU	Y	SELF EMPLOYED	SEXUAL	2009	109	2013	495	25 - 60 MONTHS	4	Y	25- 60 MONTHS	YES	PAST	PL-	PL-	-	CONJ. MICROVASCULOPATHY
38	PD	379/2011	41	M	HINDU	Y	COOLIE	SEXUAL	2011	199	2013	591	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
39	M	210/2010	32	F	HINDU	Y	HOUSEWIFE	SEXUAL	2010	120	2013	485	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
40	S	446/2013	42	M	HINDU	Y	COOLIE	SEXUAL	2013	1791	2013	1916	< 12 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6		
41	R	591/2012	33	M	HINDU	Y	COOLIE	SEXUAL	2012	176	2013	273	13 - 24 MONTHS	1	Y	13- 24 MONTHS	YES	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
42	V	116/2010	41	M	CHRISTIAN	Y	DAILY WAGER	SEXUAL	2010	149	2013	454	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
43	N	331/2011	36	M	HINDU	Y	DAILY WAGER	SEXUAL	2011	120	2013	210	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
44	D	796/2013	30	F	HINDU	Y	HOUSEWIFE	SEXUAL	2013	307	2013	307	< 12 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY

45	G	272/2012	39	M	CHRISTIAN	Y	WELDER	SEXUAL	2012	300	2013	510	13 - 24 MONTHS	2	Y	13- 24 MONTHS	YES	NIL	6/6	6/6	-	-
46	B	290/2010	46	F	HINDU	Y	DAILY WAGER	SEXUAL	2010	504	2013	532	25 - 60 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
47	K	283/2010	36	F	HINDU	Y	FLOWER SELLER	SEXUAL	2010	201	2013	985	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
48	K	485/2012	40	M	HINDU	Y	DRIVER	SEXUAL	2012	142	2013	524	13 - 24 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
49	F	562/2011	37	M	CHRISTIAN	Y	DAILY WAGER	SEXUAL	2011	210	2013	360	25 - 60 MONTHS	4	Y	25- 60 MONTHS	NO	NIL	6/60	6/6	-	VIRAL KERATITIS
50	T	280/2010	46	M	HINDU	Y	ELECTRICIAN	SEXUAL	2010	218	2013	605	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
51	S	1052/2011	50	M	HINDU	Y	SELF EMPLOYED	SEXUAL	2008	835	2013	526	25 - 60 MONTHS	1	N	NO	NO	NIL	6/6	6/6		
52	M	249/2010	55	M	HINDU	Y	DAILY WAGER	SEXUAL	2005	962	2013	835	> 60 MONTHS	1	Y	> 60 MONTHS	NO	NIL	6/12	6/12	-	CONJ. MICROVASCULOPATHY
53	V	494/2012	45	F	HINDU	Y	HOUSEWIFE	SEXUAL	2012	168	2013	573	13 - 24 MONTHS	1	Y	13- 24 MONTHS	YES	NIL	6/6	6/6	BLEPHARITIS	-
54	M	59/2009	38	M	HINDU	Y	DRIVER	SEXUAL	2009	209	2013	562	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
55	V	253/2010	32	F	HINDU	Y	HOUSEWIFE	SEXUAL	2010	215	2013	375	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	BLEPHARITIS	
56	G	1472/2013	38	F	HINDU	Y	HOUSEWIFE	BLOOD TRANSFUSION	2013	717	2013	780	< 12 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
57	R	762/2013	44	M	HINDU	Y	DAILY WAGER	SEXUAL	2013	273	2013	273	< 12 MONTHS	3	Y	< 12 MONTHS	NO	CURRENT	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
58	G	94/2009	47	F	HINDU	Y	DAILY WAGER	SEXUAL	2009	151	2013	513	25 - 60 MONTHS	3	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY
59	Y	221/2010	40	M	HINDU	Y	FISHERMAN	SEXUAL	2009	127	2013	125	25 - 60 MONTHS	2	Y	25- 60 MONTHS	YES	CURRENT	6/6	6/6	BLEPHARITIS	ALLERGIC CONJUNCTIVITIS, CONJ MICROVASCULOPATHY

60	B	651/2012	38	F	HINDU	Y	SELF EMPLOYED	SEXUAL	2012	380	2013	314	13 - 24 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6	-	ALLERGIC CONJUNCTIVITIS
61	L	1700/2013	34	M	HINDU	Y	CLERK	SEXUAL	2013	360	2013	360	< 12 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	-
62	V	1555/2013	43	M	HINDU	Y	DAILY WAGER	SEXUAL	2013	525	2013	525	< 12 MONTHS	1	N	NO	NO	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
63	M	186/2010	35	M	HINDU	Y	CUSTOMS	SEXUAL	2010	92	2013	585	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
64	B	303/2005	31	F	HINDU	Y	HOUSEWIFE	SEXUAL	2005	325	2013	570	> 60 MONTHS	1	Y	> 60 MONTHS	YES	NIL	6/6	6/6	STYE	-
65	R	689/2012	42	M	HINDU	Y	SELF EMPLOYED	SEXUAL	2012	217	2013	217	13 - 24 MONTHS	4	Y	13- 24 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
66	S	647/2013	44	F	HINDU	Y	HOUSEWIFE	SEXUAL	2012	844	2013	1123	13 - 24 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
67	S	713/2013	49	M	HINDU	Y	DRIVER	SEXUAL	2007	101	2013	803	> 60 MONTHS	1	Y	> 60 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY
68	M	435/2004	52	M	HINDU	Y	PAINTER	SEXUAL	2004	153	2013	462	> 60 MONTHS	1	Y	> 60 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY
69	S	705/2013	40	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	1106	2013	1043	25 - 60 MONTHS	3	Y	25- 60 MONTHS	YES	NIL	6/36	6/36	-	CONJ. MICROVASCULOPATHY
70	PK	371/2011	44	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	63	2013	327	25 - 60 MONTHS	3	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
71	M	571/2012	33	F	HINDU	Y	HOUSEWIFE	SEXUAL	2008	492	2013	877	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
72	T	708/2013	52	M	HINDU	Y	ATTENDER	SEXUAL	2007	79	2013	623	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/12	6/12	-	CONJ. MICROVASCULOPATHY
73	SK	292/2010	30	M	HINDU	N	SECURITY GUARD	SEXUAL/IV DRUG ABUSE	2010	198	2013	312	25 - 60 MONTHS	1	Y	25- 60 MONTHS	YES	PAST	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
74	P	405/2011	55	F	HINDU	Y	FRUIT SELLER	SEXUAL	2011	171	2013	750	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	-	CONJ. MICROVASCULOPATHY

75	M	598/2013	32	F	HINDU	Y	COOLIE	SEXUAL	2012	114	2013	146	13 - 24 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
76	R	1563/2013	37	M	HINDU	Y	COOLIE	SEXUAL	2013	178	2013	178	< 12 MONTHS	1	Y	< 12 MONTHS	NO	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
77	L	949/2011	35	F	HINDU	Y	DAILY WAGER	SEXUAL	2011	759	2013	1045	25 - 60 MONTHS	1	N	NO	YES	NIL	6/36	6/36	BLEPHARITIS	CONJ. MICROVASCULOPATHY
78	S	645/2012	56	M	CHRISTIAN	Y	DAILY WAGER	SEXUAL	2012	153	2013	248	13 - 24 MONTHS	3	Y	13- 24 MONTHS	NO	PAST	6/6	6/6		
79	P	48/2009	36	M	HINDU	Y	DAILY WAGER	BLOOD TRANSFUSION	2009	252	2013	542	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
80	VM	759/2013	42	M	HINDU	Y	COOLIE	SEXUAL	2010	169	2013	165	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/60	6/60	BLEPHARITIS	CONJ. MICROVASCULOPATHY
81	UP	464/2010	30	M	HINDU	Y	PLUMBER	SEXUAL	2010	632	2013	690	25 - 60 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
82	AM	1184/2012	7	F	HINDU	N	SCHOOL	MTC	2012	711	2013	425	13 - 24 MONTHS	1	Y	< 12 MONTHS	YES	NIL	6/6	6/6	BLEPHARITIS	
83	ABR	269/2010	45	M	HINDU	Y	MANUAL WORK	SEXUAL	2010	249	2013	658	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
84	YD	767/2013	32	M	HINDU	Y	COOLIE	SEXUAL	2013	200	2013	200	< 12 MONTHS	4	Y	< 12 MONTHS	YES	NIL	6/6	6/6	BLEPHARITIS	CONJ. MICROVASCULOPATHY
85	M	1162/2013	39	M	HINDU	Y	SELF EMPLOYED	SEXUAL	2013	100	2013	100	< 12 MONTHS	4	Y	< 12 MONTHS	YES	NIL	6/6	PL+	-	CONJ. MICROVASCULOPATHY
86	M	840/2013	40	F	HINDU	Y	FLOWER SELLER	SEXUAL	2009	709	2013	355	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
87	P	1173/2009	55	F	HINDU	Y	HOUSEWIFE	SEXUAL	2009	250	2013	400	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/24	6/24	-	-
88	S	676/2010	30	F	HINDU	Y	COOLIE	SEXUAL	2010	348	2013	500	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	-	-
89	M	540/2011	35	M	HINDU	Y	PAINTER	SEXUAL	2011	180	2013	457	25 - 60 MONTHS	2	y	25- 60 MONTHS	YES	PAST	6/6	6/12	-	-
90	N	608/2012	14	M	HINDU	N	STUDENT	MTC	2013	318	2013	318	< 12 MONTHS	2	y	< 12 MONTHS	YES	NIL	6/6	6/6	MOLLUSCUM CONTAGIOSUM	-

91	S	444/2008	30	F	HINDU	Y	SWEEPER	SEXUAL	2008	350	2013	435	25 - 60 MONTHS	1	Y	25- 60 MONTHS	YES	NIL	6/6	6/6	-	ALLERGIC CONJUNCTIVITS
92	E	539/2011	38	M	HINDU	Y	DAILY WAGER	SEXUAL	2011	335	2013	534	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6		
93	J	294/2010	41	M	CHRISTIAN	Y	SELF EMPLOYED	SEXUAL	2010	234	2013	398	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	NIL	6/6	6/6	BLEPHARITIS	-
94	JM	699/2012	33	M	HINDU	Y	COOLIE	SEXUAL	2012	342	2013	467	13 - 24 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6		
95	LM	709/2012	60	M	HINDU	Y	UNEMPLOYED	SEXUAL	2012	319	2013	410	13 - 24 MONTHS	1	Y	13- 24 MONTHS	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATHY
96	P	235/2011	33	F	HINDU	Y	HOUSEWIFE	SEXUAL	2011	270	2013	390	25 - 60 MONTHS	1	Y	25- 60 MONTHS	NO	PAST	6/6	6/6	-	COMPLICATED CATARACT
97	K	1556/2013	34	M	HINDU	Y	MANAGER	SEXUAL	2013	400	2013	400	< 12 MONTHS	1	N	NO	YES	NL	6/6	6/6	RECURRENT STYE	-
98	S	1403/2012	30	F	HINDU	Y	COOLIE	SEXUAL	2012	780	2013	759	13 - 24 MONTHS	1	N	NO	YES	NIL	6/6	6/6	BLEPHARITIS	-
99	D	894/2012	40	M	HINDU	Y	PLUMBER	SEXUAL	2012	338	2013	650	13 - 24 MONTHS	1	Y	13- 24 MONTHS	YES	NIL	6/6	6/6		
100	T	1709/2013	33	F	HINDU	Y	SWEEPER	SEXUAL	2013	560	2013	560	< 12 MONTHS	1	N	NO	NO	NIL	6/6	6/6	-	CONJ. MICROVASCULOPATH

OCULAR FINDINGS							
RIGHT EYE			LEFT EYE				
POSTERIOR SEGMENT	NEURO OPHTHAL	ORBIT	ADNEXA	ANTERIOR SEGMENT	POSTERIOR SEGMENT	NEURO OPHTHAL	ORBIT
ACUTE RETINAL NECROSIS	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	ACUTE RETINAL NECROSIS	-	-
CMV RETINITIS BURNT OUT	-	-	-	-	CMV RETINITIS BURNT OUT	-	-
CMV RETINITIS BURNT OUT	-	-	-	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	MULTIPLE CRANIAL NERVE PALSY	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
TOXOPLASMA RETINITIS , VITRITIS	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY , INFLAMMATORY GLAUCOMA	TOXOPLASMA RETINITIS , VITRITIS	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	ALLERGIC CONJUNCTIVITIS, CONJ MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	NON HEALING FUNGAL CORNEAL ULCER	-	-	-

-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
RETINITIS	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	RETINITIS	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	ALLERGIC CONJUNCTIVITIS	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-
-	-	-		CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-

-	-	-	-	ALLERGIC CONJUNCTIVITIS, CONJ MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	MOLLUSCUM CONTAGIOSU M	-	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
TOXO PLASMA RETINITIS	-	-	-	CONJ. MICROVASCULO PATHY	TOXO PLASMA RETINITIS	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-

-	-	-	HEALED HERPES	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	ALLERGIC CONJUNCTIVITIS, CONJ MICROVASCULO PATHY	-	-	-

-	-	-	-	ALLERGIC CONJUNCTIVITIS	-	-	-
HIV RETINOPATHY	-	-	-	-	HIV RETINOPATH	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	-	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	HIV RETINOPATH Y	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY, OSSN	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
RETINAL VASCULITIS	DISC PALLOR	-	-	CONJ. MICROVASCULO PATHY	RETINAL VASCULITIS	DISC PALLOR	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-

-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
INTERMEDIATE UVEITIS	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	INTERMEDIA TE UVEITIS	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	-	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS		-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	-	-	BLEPHARITIS	CONJ. MICROVASCULO PATHY	-	-	-
-	DISC PALLOR ,NASAL FIELD DEFECT	-	-	-	-	DISC PALLOR , OPTIC NEURITIS	-
HIV RETINOPATHY	-	-	-	-	HIV RETINOPATH Y	-	-
HIV RETINOPATHY	-	-	-	-	HIV RETINOPATH Y	-	-
-	-	-	HERPES ZOSTER	-	-	-	-
-	-	-	MOLLUSCUM CONTAGIOSU M	-	-	-	-

-	-	-		ALLERGIC CONJUNCTIVITS	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-
-	-	-	CONJ. MICROVASC ULOPATHY	-	-	-	-
-	-	-	COMPLICATE D CATARACT	-	-	-	-
-	-	-	RECURRENT STYE	-	-	-	-
-	-	-	BLEPHARITIS	-	-	-	-
-	-	-	CONJ. MICROVASC	-	-	-	-